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EPOCH-MAKING BOOKS IN BRITISH SURGERY.

BY SIR D'ARCY POWER, K.B.E., LONDON.

V. THE SURGEONS MATE BY JOHN WOODALL.

THE interest of this book is twofold; first on account of the author, and secondly because of the subject on which he wrote. The barber surgeons set their house in order in 1540, and organized a system of teaching and examination for civil practitioners. Gale and Clowes sought to instruct military surgeons in their duties. It was not until Woodall published "The Surgeons Mate" in 1617 that any thought was given to the ship's surgeon. Woodall, after serving abroad, was elected surgeon to St. Bartholomew's Hospital in 1616, the year in which his colleague Dr. William Harvey was delivering his first course of lectures on the circulation of the blood. Woodall was appointed the first Surgeon-General when the East India Company was formed into a joint stock business. In this position he must soon have seen the need of some official text-book for the guidance of the younger medical officers in the service. The Governors of the Company also recognized the want, empowered him to fill it, and generously undertook to defray the cost of a book to be published under their authority. The result was: (1) A series of notes on the contents of the chest with which every medical officer in the navy and army was obliged to furnish himself. The notes consisted of a description of the drugs, instruments, and appliances contained in the chest —illustrated by plates to prevent the possibility of any mistake. (2) The little guide must have been successful, and in 1628 Woodall published the "Viaticum" or "Pathway to the Surgeons Chest," which contained the general principles of the treatment of wounds. (3) A volume of Military and Domestic Surgery, consisting of a revised version of "The Surgeons Mate" and the "Viaticum" with many alterations and additions. This was published in 1639, when, as he says, "time hath overtaken me having run through the cares of sixty-nine years, old age being an enemy to study, for my sight being weakened, my memory much impaired and my capacity utterly unable to perform so hard a task". Indeed, it is saddening to read the book. The

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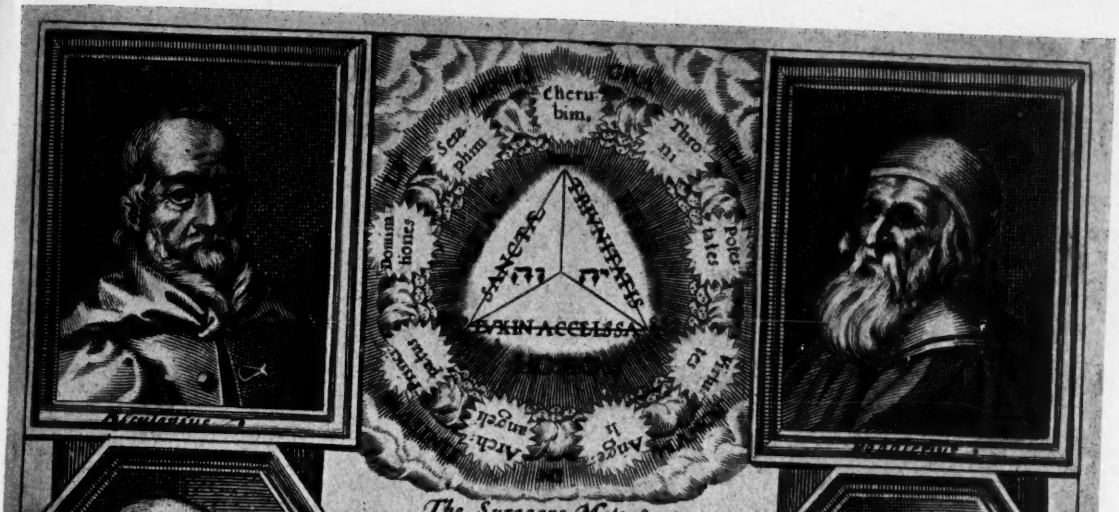
earlier parts are well and clearly written, giving dogmatic accounts of Woodall's own experience : the later additions show that he has become lost in mysticism and alchemy. He relies upon the experience of others rather than upon his own, and describes in doggerel verse what he should have said in prose.

The Surgeons Mate was written for use "on a great Ship upon a long voyage for sea service as suppose for an East India voyage". It deals entirely with the diseases and injuries of seamen, for, as Woodall says, "women in long voyages are rare creatures". The advice offered to the Surgeon's Mates, or Assistant Surgeons as they would be called nowadays, is thoroughly practical. The duty of the Surgeon's Mate is to behave to his chief surgeon "wisely, lovingly and diligently ; and in many respects he is obliged to his Mate as the wife to her husband for he ought to be in every point ready to obey his reasonable will, I mean only in things concerning his calling ; dutiful, diligent, willing, careful, secret and loving, yea to show his love in covering his Mate's weakness. He ought to be gentle and kind in speech and actions towards all ; pitiful to them that are diseased and diligent in ministering to them such fitting remedies as he shall receive or be directed to give from his Mate, but none without his order, for that the Surgeon's order is his discharge or warrant whatever happen. Yea, let him privately lend his best advice to his Mate, the prime surgeon, on every needful occasion, kindly, submissively and lovingly ever as under correction, for if comparisons or oppositions take place betwixt them it is not only dangerous to all the ship's company but also a disgrace to themselves and a great scandal to their calling for avoyding which it is fit that the younger obey the elder. . . . It showeth an excellent thing in a young man to please an honest, quiet man but to live in love with an ambitious or otherwise a froward, vicious man showeth wisdom in the sufferer." The advice is excellent for those who were thrown into intimate contact with each other during the many long months of a voyage to the East Indies round the Cape of Good Hope, badly housed, worse fed, in calms and in storms. It was perhaps worth while to give it, but in most cases it must have been a counsel of perfection.

"The Surgeon's Mate ought daily to visit the cabins of the men to see who hath any sickness or imperfection. Yea, and the Chirurgeon ought morning and evening to seek for poor men in their cabins, or as soon as they are missing at their Messes and inquire for them and see that their cabins be sweet and their provisions suitable, or to move and intreat the Master or Governor of the Ship for redress in such cases for fear of a general infection. Likewise his care must be to keep all the instruments clean from rusting and to set his lancets and razors as oft as need be. It may be that he will say to himself ; it is a base office belonging to mere Barbers and Grinders. I never give my mind to it &c. But let me friendly tell the Surgeon's Mate it is to the credit of a young artist to take a vein smoothly and neatly, as also to shave well is praiseworthy. Whereof I wish him to practise to do it and to be ever learning, for I am sure he shall never know half he ought to know though he do his best."

Woodall's teaching, as might be expected, extends both to medicine and surgery. On the medical side constipation of the most severe character was an ever present trouble, and for its cure clysters and mechanical means of



The Surgeons Mate
or

Military & Domestique Surgery.

Discouering faithfully & plainly ye method and order of ye Surgeons chest, ye vse of ye Instruments, ye vertues and Operations of ye Medicines, wth ye exact Cure of wounds made by Gunshott, and otherwise, as

Namely:

Wounds, Apostumes, Vlcers, Fistula's, Fractures, dislocations, wth ye most easie & safest wayes of Amputation or dismembring, The cures of the Scurucy, of ye fluxes of ye bellie, of ye Collicke and Ilhaca Passio, , of Tenasmus and Exitus Ani, and of the Calenture, with

A Treatise of ye cure of ye Plague.

Published for the seruice of his Maᵗⁱᵉ and of the com: wealth.

By

John Woodall
Mr in Chyrurgerie.

LONDON Printed by Rob: Young, for Nicholas Bourne, and are to be sold at his Shop at the south entrance of the Royall Exchange. MDCXXXIX.

relief are recommended to the exclusion of purgatives, of which Woodall seems to have had an especial abhorrence. Diarrhœa—fluxes, as he calls them —were also of common occurrence. He had no means of discriminating them, so he groups them roughly into Lienteries, Diarrhœas, and Dysenteries. For them " Laudanum is the only sure help and if at first it cure not, yet it giveth rest, whereby the party afterwards will be the stronger to endure other reasonable medicine, always remembering there be three or four hours betwixt each dose ". " And among other needful instruments for poor sea-men in fluxes never be unfurnished in the ship of one or two close-stools with doors to them and brass pails, that poor miserable men in their weakness may be eased thereon and not be constrained to go either to the beake-head, or shrouds (as they term it) for that not only increaseth the disease but also causeth the falling down of the Ano, a fearful accident."

Of scurvy Woodall says, " some men conceive this disease happeneth to sea-men only, through long being at sea without touch of land, as is seen in the East India voyages ; our men have it betwixt England and the Cape *de bon speranze* ". It was so common indeed that " A fever at sea commonly ends in the Scurvy, wherefore by the way beware of too large purging or phlebotomy which increase oft the grief and makes it incurable. I speak this because I have noted there is a fault in young Surgeons of forwardness in taking too much blood at Sea."

The best-known passage of Woodall's writings occurs in his treatment of scurvy, where he says: " The Chirurgeon or his Mate must not fail to persuade the Governor or Purser in all places where they touch in the Indies, and may have it, to provide themselves of juice of Oranges, limes, or lemons and at Banthame of Tamarinds, for these good helps which you shall find in the Indies do far exceed any that can be carried thither from England, and yet there is a good quantity of Juice of Lemons sent in each ship out of England by the great care of the merchants, and intended only for the relief of every poor man in his need, which is an admirable comfort to poor men in that disease. Also I find we have many good things that heal the Scurvy well at land, but the Sea Chirurgeon shall do little good at Sea with them."

Woodall's surgical treatment is excellent, for it is simple and is the outcome of his personal experience abroad and at home. Speaking of the treatment of fractures he says " it is manifest that in simple fractures the placing of the bones and keeping them so is the most work of the Cure and nothing cureth a fractured bone so much as rest, wherefore when a bone is newly placed and shall be troubled with much lifting and rowling [bandaging] it cannot but hinder union."

Of dislocations he says, wisely enough, " When the Surgeon taketh a view of a member dislocated it were good that the other side also were uncovered, that thereby the true form and situation of the diseased limb being well regarded and compared together with the whole joint the better judgement and truer indication might be taken. I mean if one shoulder or elbow be out of joint let the Surgeon make bare the other side also. Then seek to reduce or place the bone by those means which in thy own reason seem fittest." For his own part Woodall preferred to reduce dislocations of the shoulder by an instrument " which I learned the practice of in Poland whilst

I lived there. . . . I may truly say I have set with it above 500 joints at times". It consisted of an oval-headed wooden rod which fitted into the axilla and acted on the same principle as the more usual method of reduction by the heel in the axilla.

He advocates free drainage of gunshot wounds in the following words: "But ever take heed to avoid the old received error of unwise practitioners whose use is to cram the wounds, be they incised wounds or contused wounds, full of bole or restringent powder, or some other stuff and then thrust in pledgets or dossels into each corner of the wound, yea sometime forgetting to take all out at the next dressing yet think they have done all workmanlike and very artificially, not considering the harm that often ensueth thereby."

The farewell appended to his last treatise shows Woodall to have been an honest, strict, but not dogmatic Puritan who endeavoured to serve his generation to the very best of his ability. It runs: "Loving Brethren, if the meaness of my style, the weakness of my aged memory, capacity or what else soever is not answerable to thy expectation from me, yet take it in good part I pray thee, as proceeding from a Friend, who, if his talent could have afforded better, would willingly have imparted it to thee. The former was the fruits of my youth, this, such as the weakness of old age can afford. The public good acceptance of the former hath purchased the latter. Vale in Christo."

The portrait by G. Glover in the lower central panel of the title page is clearly a speaking likeness. George Glover (fl. 1625–1650) was one of the earliest and most successful engravers of his day. He used his graver boldly and effectively, and was especially noted for his portraits. The frontispiece, here copied from the 1630 edition of "The Surgeons Mate," is a good example of his work.

INVASION OF THE SKULL BY DURAL TUMOURS.

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THE meningeal tumours that form the subject of this communication have been recognized for very many years, and have been variously named according to the changing conceptions of their pathogenesis and nature. Thus Virchow[1] considered that they were of two kinds, naming these respectively 'sarcoma' and 'psammoma', the latter title being assigned to those growths that contain the small calcified 'psammoma bodies'. But he confesses the difficulty of distinguishing some of the sarcomas from fibromas, and also of assigning the name 'sarcoma' to growths that are not characterized by the type of malignancy that it usually connotes. He states definitely, however, that there are undoubted dural sarcomas and that these may result in a large growth of bone on the surfaces of the calvarium. Later authors have for the most part used the expression 'endothelioma' to designate all types—that is, the apparently purely dural, and those associated with bony overgrowth—the presence of psammoma bodies being noted in many examples of all types. More recently the term 'meningioma' suggested by Cushing[2] has received almost universal acceptance as comprising two main classes, named by him the *spherical*, with small dural attachment, and the *plaque-like*, with extensive involvement of the membrane. It is clear, however, that though most observers, with the tremendous exception of Cushing, must confine their investigations to isolated examples, all are concerned with growths of the same class, as is shown by the constant and collectively individual characters exhibited.

THE VARIETIES AND NATURE OF DURAL TUMOUR.

A dural tumour is the boon that a surgeon usually asks when he opens the head for tumour, and their characters will not be detailed here otherwise than to note the well-known facts that the plaque-like type commonly results in the development of a large overlying bony mass that has received the long but non-committal name of *hyperostosis*, while the more spherical variety with small dural attachment does not produce in the calvarium anything so striking.

Since Schmidt[3] in 1902 expounded their possible arachnoid origin, much has been written of them, and more since Cushing and his fellow-workers gave out the results of their research and great experience. The views of Cushing's school are accepted by practically all authors since 1922, and by many before that year, and his statement of their purport[4] is therefore taken by the writer as a starting-point.

"The endotheliomas must of course be regarded as benign tumours, but it is known that the growth tends to insinuate itself into crevices and may surround and incorporate important anatomic structures like the blood-vessels of the cranial

base without damaging them. In view of this it might be assumed that under the influence of intracranial tension, the tumour cells in the process of their multiplication become crowded into and through the vascular dural spaces, and finally into the canaliculi of the bone. In consequence of this the bone becomes irritated with subsequent osteoblastic proliferation which provokes the hyperostosis. There can be little doubt that the thickening occurs in this way, but intracranial tension can have nothing to do with it in view of the fact that the flat endotheliomas which do not increase tension are, as we have seen, those which most often tend to invade bone. There must be some other reason, therefore, to account for this peculiar process. One occasionally finds a meningeal endothelioma in which there is a tendency to bone formation within the tumour itself, and therefore the process may bear some relation to the bone-forming properties of the meninges, but without further data on the subject, speculation is futile."

Now a benign infiltration cannot be regarded as a pathological commonplace, and these words undoubtedly contemplate the existence of a peculiar neoplasm not paralleled in any other bodily process, a benign growth with malignant habits. Such a conception appears to govern all recent writings, with the possible exception of Phemister's[5], and consequently stress is laid upon the normal proliferation of the arachnoid into the substance of the dura,[3, 6, 7] this being held to compare with the peculiar infiltration of the skull seen in the neoplasm. It is our present purpose here to examine how invasion of the skull by dural tumours differs from that of other bones by known malignant growths. The results of this examination suggest that a difference does not exist, and that once it is recognized as malignant—as indeed it was in certain instances by Virchow—the growth of these tumours takes its place naturally among the ordinary pathological processes. A somewhat similar investigation was published in 1923 by Phemister,[5] neither the findings nor conclusions of which are in agreement with those presently to be described, but the writer regrets that this paper had escaped his attention until his own was nearly complete.

AN INVESTIGATION OF TWO DURAL TUMOURS.

Methods of Investigation.—Information was obtained as to the spread of dural and known malignant tumours in bone by macroscopic, skiagraphic, and microscopical examination. Published microscopical examinations are many, and for the most part their findings are in agreement; but the skiagraphic appearances both of dural and of unquestionably malignant tumours invading bone are mostly incomplete and nowhere minute, and the same is to a less extent true of macroscopic features. Further, only Phemister really attempts to correlate the skiagraphic and the other findings.

A Plaque-like Tumour.—The first specimen investigated is a typical parasagittal dural tumour of the plaque type with great extracranial bony development, removed, the writer confesses at once, in a way that he had not intended—that is, post mortem. It has been divided in the coronal plane, and is seen to involve (*Fig.* 1) both inner and outer aspects of the skull and the dura mater. The shape of the bony swelling is roughly a truncated cone, but the extreme periphery rises gradually from the surface of the skull, the slope changing to a steep ascent as the middle of the growth is approached.

The bony tissue at the periphery consists of dense bone of uniform consistence, but the main central mass of the tumour is composed of alternating bony and soft strands lying radially to the curved surface of the calvarium. The outline of the calvarium can be followed distinctly right across the section. On its inner aspect a similar arrangement is seen, but the bony spicules are finer. The dura is traced right across the section, and on its inner surface is yellow nodular growth which fails to cross the falx, whereas the bony growths straddle the middle line. In the calvarium itself, patchy condensation and destruction of the bone is seen, in the interstices of which the diploë is replaced by growth in which in places there is hæmorrhage. The radial arrangement stops short exactly at the two surfaces of the skull. Four zones are thus seen in *Fig.* 1:—(A) the intradural, uncalcified, (B) the extradural, ossified, (C) the cranial, (D) the extracranial, ossified—separated from one

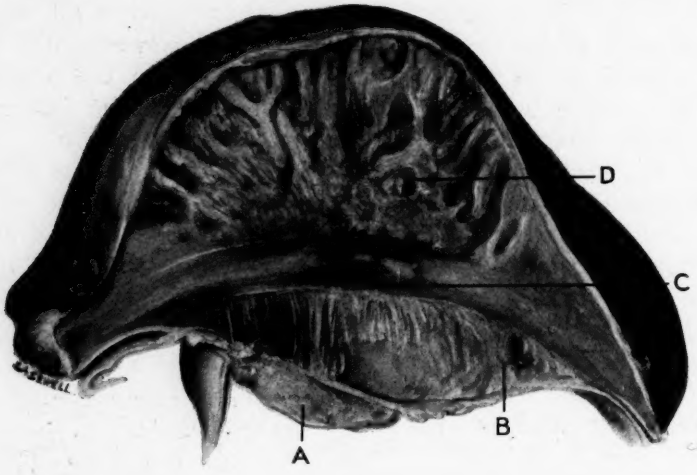

FIG. 1.—A dural tumour of the plaque type. Zone A, intradural; B, extradural; C, cranial; D, extracranial.

another by the natural anatomical boundaries of the dura and the outer and inner surfaces of the skull.

Skiagrams.—That taken during life (*Fig.* 2) shows well the striate appearance; but that of a slice of the tumour (*Fig.* 3) shows in addition that the apparently uniform part of the ossification has a complex structure. It is really arranged in laminæ laid down concentrically with the curvature of the calvarium both on the outer and the inner surfaces. Crossing these concentric laminæ are radial darker lines that become more definite and wider as the middle of the growth is approached. In contradistinction from the lighter bony lines these darker ones in some cases pass through the calvarium, and it will be shown that they represent canals in the bone containing vessels surrounded by growth, such vascular paths constituting the routes by which the growth spreads. The sharp distinction between the ossified extradural zone (*Fig.* 1, B), and the non-ossified intradural (*Fig.* 1, A) is also seen. The cranial zone (*Fig.* 1, C) shows also effects of invasion by the tumour. In the

extreme periphery of the removed portion of bone the diploic spaces are seen. In the region of the tumour the diploë has disappeared to a large extent, the bone of the calvarium appearing solid and very dense. In the middle of the tumour, however, there was a patchy destruction of bone, as shown

FIG. 2.—Skiagram taken during life of the skull from which the specimen shown in *Fig.* 1 was taken.

by irregularly arranged spaces in the bony shadow. In addition the dark lines already said to represent vessels can be seen passing right across the thickness of the calvarium into the extracranial zone (*Fig.* 1, D).

FIG. 3.—Skiagram of slice of the specimen shown in *Fig.* 1. Complex structure of bony deposit.

Microscopical Appearances.—These preparations call for some apology for their imperfections. The method of bulk hardening necessitated by the scheme of investigation does not result in good fixation, and also, even after

prolonged decalcification, the tissue was abominably hard. *Fig.* 4 shows the soft intradural zone (*Fig.* 1, A), where the typical spindle cells sometimes arranged in whorls are seen, the appearance that led Virchow to describe these tumours as sarcomas. *Fig.* 5 shows the periphery of the extracranial zone,

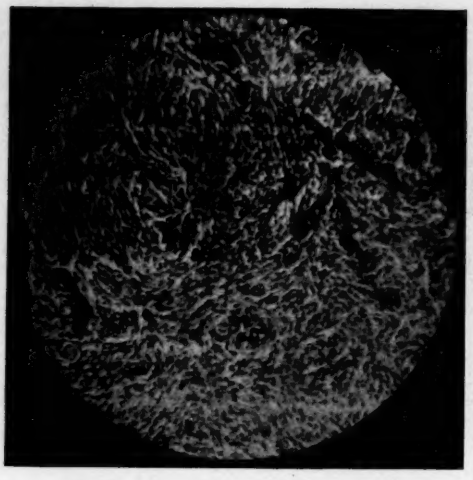

FIG. 4.—Section of Zone A, (*Fig.* 1). Typical appearance of dural tumour. (Low power.)

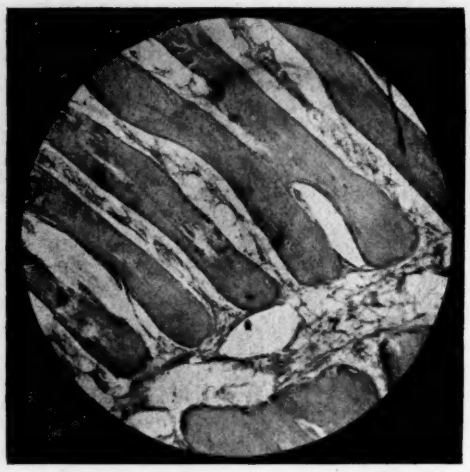

FIG. 5.—Section of peripheral part of Zone D, (*Fig.* 1). Concentric lamination. Transversely lying vessel surrounded by tumour cells. (Low power.)

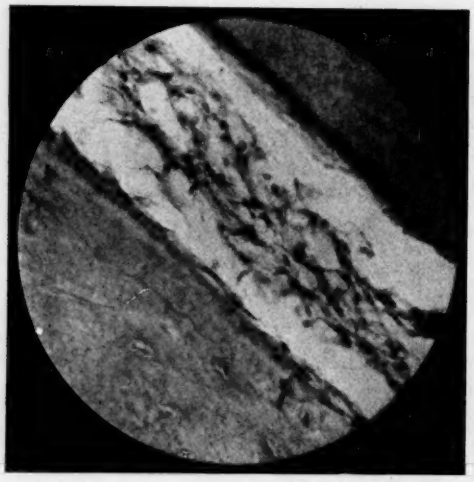

FIG. 6.—Section of one of the concentric spaces and adjacent laminæ of *Fig.* 5. Tumour cells in the vascular space. Bone cells along the walls. (High power.)

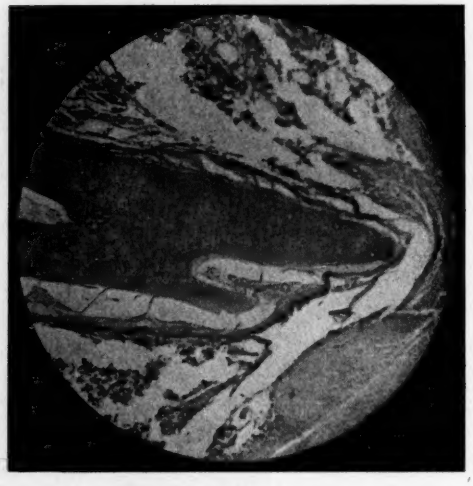

FIG. 7.—Section of a spicule from Zone B, (*Fig.* 1) in contact with the dura. (Low power.)

where the concentric laminæ are well seen in section. Between these, in spaces looking like Haversian canals, are vessels and small deposits of growth. Crossing them at nearly a right angle, at one place a large vascular space is seen containing a vessel surrounded by growth. This corresponds to one of the dark

radial lines seen in the skiagram. A higher magnification (*Fig.* 6) shows growth in a concentric bone space, the walls of which are lined by bone cells. These appearances are identical with those described by most authors. *Fig.* 7 shows the point of a spicule from Zone B, (*Fig.* 1) with active bone

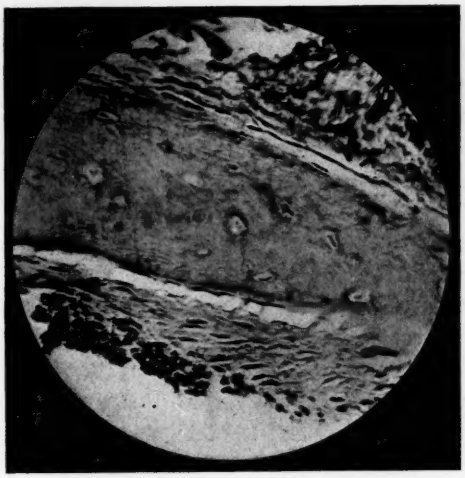

FIG. 8.—Higher magnification of the spicule shown in *Fig.* 7. The spicule has bone cells in contact with it and tumour cells alongside these.

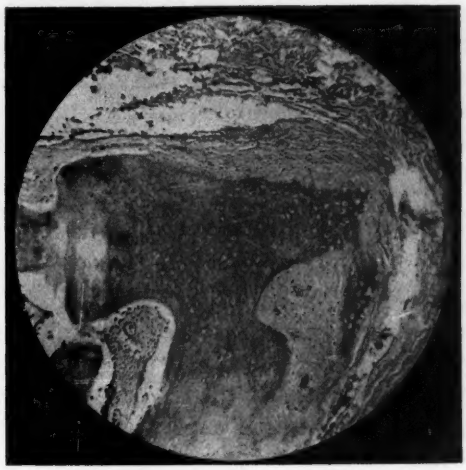

FIG. 9.—Section of the edge of Zone D, (*Fig.* 1) near the summit. Active bone formation. Incompletely calcified, swollen, and numerous osteoblasts. (High power.)

formation in contact with the outer surface of the dura, which is visible and does not appear to be invaded at this point. Higher magnification of the same section (*Fig.* 8) again shows activity of bone cells and the presence of growth alongside. *Fig.* 9 shows the point of a coarse spicule of bone from Zone D, (*Fig.* 1) immediately under the surface of the tumour, and here again there is great activity of the bone cells, as is demonstrated by the cap of incompletely calcified bone and the large numbers of swollen osteoblasts arranged along the edges of the newly-formed bone. *Fig.* 10 shows destruction of bone in the tumour mass such as is obviously necessary in the process of the substitution of the coarse radial arrangement for the concentric close stratifications.

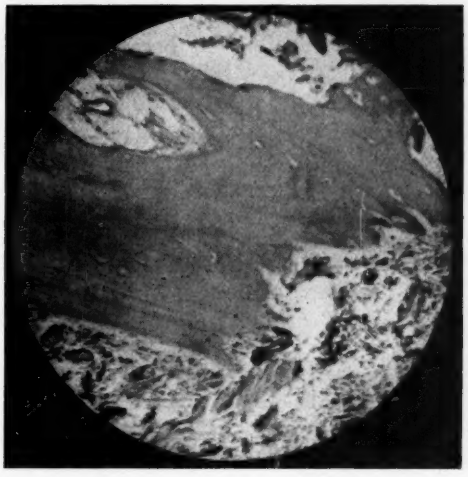

FIG. 10.—Section of middle of Zone D, (*Fig.* 1). A spicule is being destroyed, and fragments of bone in the process of destruction are visible in the tumour mass.

THE PROCESS OF INCREASE IN THIS TUMOUR AS DEDUCED FROM THE FOREGOING OBSERVATIONS.—A growth has been seen invading the skull, the first effect being the stimulation of the skull to the formation of new bone

exactly as if the stimulus had been an inflammatory one. Thus has been produced a peripheral mass of dense bone rising gradually from the surface of the calvarium. Subsequently the conventions of the osteoblasts have been overruled at the dictation of the tumour, so that while their activity has become far greater, it has been perverted to the formation of a structure composed of radially arranged elements. The disposition of these has been determined by the paths of the newly-formed vessels of the tumour alongside which the growth cells have spread. The rapid increase in bone formation and its alteration in character, seen at the site of change from the peripheral to the perverted type of ossification, are shown macroscopically by the curvature already described of the surface of the tumour—that is, a gradual peripheral rise changing to a steep ascent. It should thus be noted that the common statement of the majority of authors that the most active part of the tumour is the summit is a misappreciation of the observed facts, a truer conception being that the infiltrating periphery meets a powerful reaction on the part of the skull, the resulting new bone having a structure of the inflammatory type, but that when this resistance is overcome the tumour gains complete control of the activities of the osteoblasts it has perverted. A reference will be made to the possible origins of these perverted osteoblasts.

A Tumour of the Spherical Type.—*Fig.* 11 shows diagrammatically a section of such a growth, with little visible change in the overlying skull.

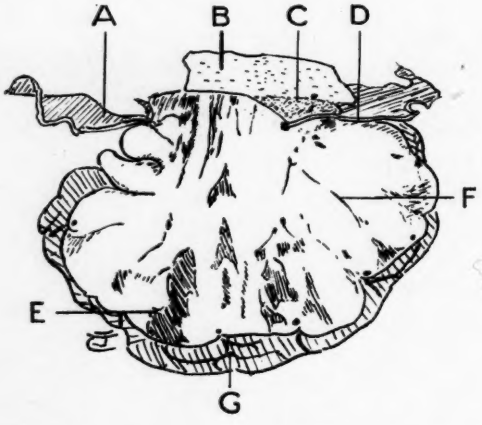

FIG. 11.—Diagram of a dural tumour of the spherical type with small pieces of attached dura and skull. A, Dura; B, Skull; C, New bone; D, Dura; E. Hæmorrhage; F, Vessel in tumour; G, Vessels on surface of tumour.

The features of lobulated surface and small area of attachment to the dura are obvious, while the only visible change in the small piece of calvarium adjacent is some irregularity at the site of attachment of the tumour and a very small deposit of new bone. This growth was pressing on the upper part of the Rolandic area and was removed successfully by Mr. Trotter, to whom the writer is indebted for the specimen. The bony change in this specimen is slighter than is often seen in this type of growth, and on one occasion the writer was present at the removal of such a tumour arising from the squamous

temporal bone, which was so greatly increased in thickness by a dense bony deposit that the site of origin was at first mistaken for the petrous.

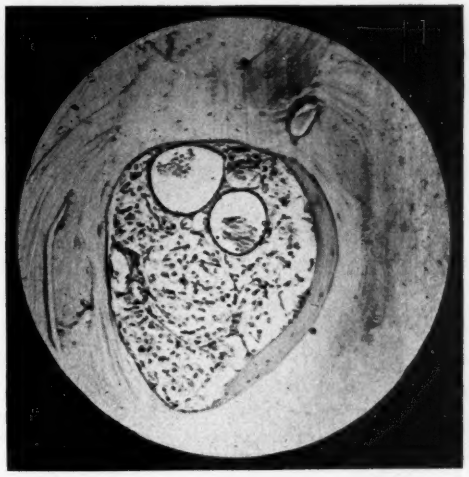

FIG. 12.—Section of the bone removed with the tumour shown in *Fig.* 11. A vascular space in the bone contains vessels and growth, and is lined by bone cells.

Microscopical Appearances.—Fig. 12 shows a vascular space in the skull overlying this growth, invaded by tumour cells lying alongside vessels. Osteoblasts line the wall of the vascular space.

COMPARISON OF THE DURAL GROWTHS WITH OTHER TUMOURS.

The Alleged Distinction between 'Tumorous' and 'Non-tumorous Bone or Hyperostosis.—So far the writer is in agreement with most published histological findings.[4, 8] But here many authors[5] draw a distinction between bone formed by a bone as the result of invasion by a tumour, such bone being called 'non-tumorous', or 'hyperostosis', and bone formed by osteoplastic growth, such bone being called 'tumorous'. The next stage of the present inquiry—that is, investigation of the periosteal sarcomas on similar lines—shows, in the writer's opinion, that this distinction is false. Kolodny, in his report of the Registry of Bone Sarcoma of the American College of Surgeons[18], is also of opinion that no histological or anatomical distinction can be made between tumorous and non-tumorous bone in sarcoma.

An Osteoplastic Periosteal Sarcoma.*—

Skiagraphic Appearances.—These have essentially the same elements as those of osteoplastic (the plaque-like type) dural tumours—that is, patchy condensation and destruction of the cancellous and the cortical bone, with

* The not entirely suitable word 'osteoplastic' is used throughout this paper to denote tumours of bones that result in new bone formation. The words 'osteogenetic' and 'osteogenic' are open to misconstruction by comparison with other medical terms. Thus 'pyogenic' signifies 'pus-forming', while 'necrogenic', in the case of the infective process known as 'verruca necrogenica', indicates not 'productive of gangrene', but 'produced by a corpse'. It is obviously of importance that there be no analogous confusion when dealing with bone-forming tumours.

the subperiosteal deposition peripherally of concentric or parallel striæ, and in the main mass of the tumour of radial striæ. *Fig.* 13 is a skiagram of a slice of a tibia of a boy, age 16, the seat of a periosteal sarcoma of very rapid growth, causing fever and intense pain. Large amounts of irregular new bone are seen in the cancellous tissue near the tumour, together with large and small patches of bony destruction. The cortex is thickened a long way below the obvious growth by the subperiosteal deposition of new bone, extending as far down as the lower third of the shaft. This bone consists of laminæ parallel with the long axis and concentric with the curvature of the surface of the shaft. At the lower edge of the main mass of the tumour, short radial striæ are seen under the concentric laminæ, which in the main tumour become coarse radial spicules.

FIG. 13.—Skiagram of a slice of a periosteal sarcoma from the tibia of a boy, age 16. Very rapid growth. The levels at which the microscopic sections shown were taken are indicated by letters A to E.

Thus, compared with the appearances of the osteoplastic dural tumours, the differences are those of extent only. The irregular changes in the cancellous tissue correspond with those seen in the diploë and calvarium. The new subperiosteal bone formation having apparently an inflammatory character is similar to that seen under the pericranium, but is very much more extensive, owing no doubt to the very rapid growth of the tumour, while the change from parallel concentric laminæ to radial striæ in the immediate vicinity and substance of the main mass is again seen to occur and to be associated with a sudden increase in the steepness of the tumour surface, which in the case of the periosteal sarcoma corresponds with the abrupt rise of the edge of the tumour mentioned in clinical descriptions of such growths.

Microscopical Appearances.—The growth is a mixed-celled sarcoma, but for the most part it is spindle-celled. As regards bone formation the appearances are essentially identical with those of the plaque-like dural tumour. *Fig.* 14 shows the cortex and periosteum at the level A in *Fig.* 13—that is, a short way above the malleolus. There is a little increased activity of the subperiosteal bone cells, but the appearances are otherwise those of normal bone. *Fig.* 15 is a section taken from the level B, an inch higher up the shaft, and here there is increased subperiosteal activity, some apparently normal bone having been laid down on the surface of the cortex, the old cortex and the new bone being visibly distinct. *Fig.* 16, a section taken from a point at level C, shows concentric parallel lamination in the process of formation. The tumour cells are also seen around the vessels that lie in their Haversian canals. *Fig.* 17 shows a higher magnification of

the same section with intense subperiosteal activity and tumour cells here and there. *Fig.* 18 shows concentric parallel laminæ well formed at the level D. As in the last section, the spread of the growth around the vessels is well seen. *Fig.* 19, a section at the level E, shows the early formation

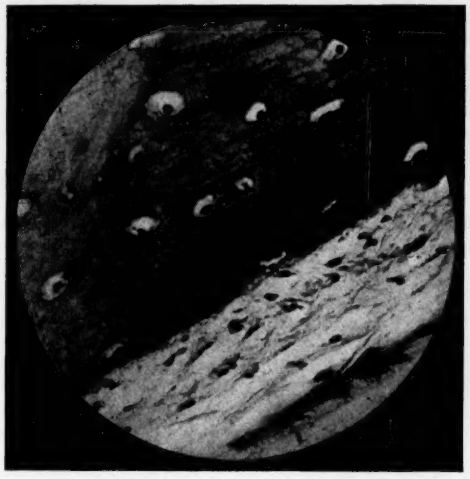

FIG. 14. — Periosteum and cortex of sarcoma at level A of *Fig.* 13. Active but almost normal bone. (High power.)

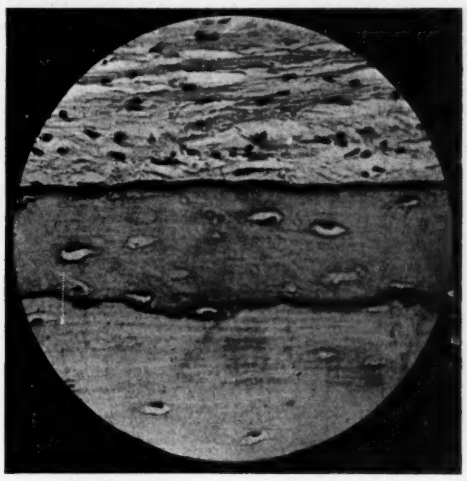

FIG. 15.—Section of periosteal sarcoma at level B of *Fig.* 13. Great activity. New bone laid down on surface of cortex. (High power.)

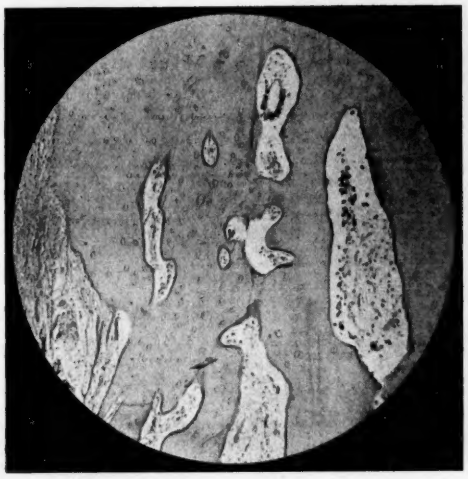

FIG. 16.—Section of periosteal sarcoma at level C of *Fig.* 13. Concentric lamination in process of formation. Growth in Haversian canals around vessels. (Low power.)

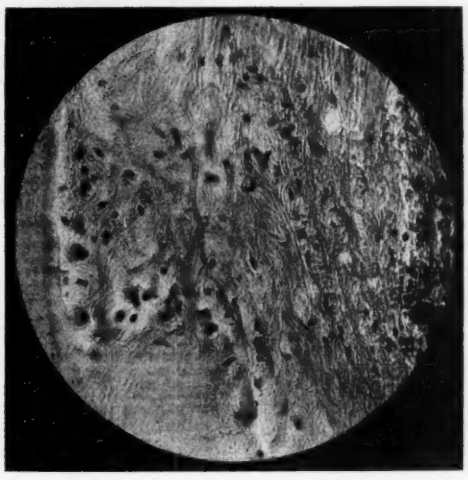

FIG. 17.—Higher magnification of *Fig.* 16. Intense activity of bone cells. Tumour cells here and there.

of the radial elements, the growth cells spreading around newly-formed vessels which are arranged radially to the surface of the old cortex. The bone formation by osteoblasts under the influence of the tumour growth is thus made up of radial spicules. *Fig.* 20 shows the edge of the main mass

of the growth. A radial bone spicule is seen in active formation by the agency of large numbers of swollen osteoblasts with tumour substance (spindle-celled sarcoma in this part of the growth) alongside.

FIG. 18.—Section of periosteal sarcoma at level D of *Fig.* 13. Well-formed concentric laminæ. Growth around vessels in the Haversian canals. (Low power).

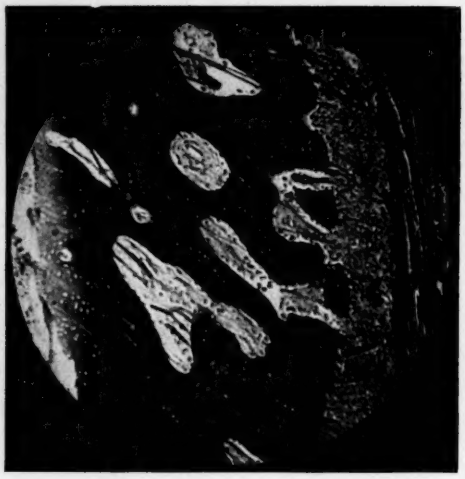

FIG. 19.—Section of periosteal sarcoma at point E of *Fig.* 13. Early formation of radial elements. The growth is spreading around vessels disposed radially to the surface of the old cortex. (Low power.)

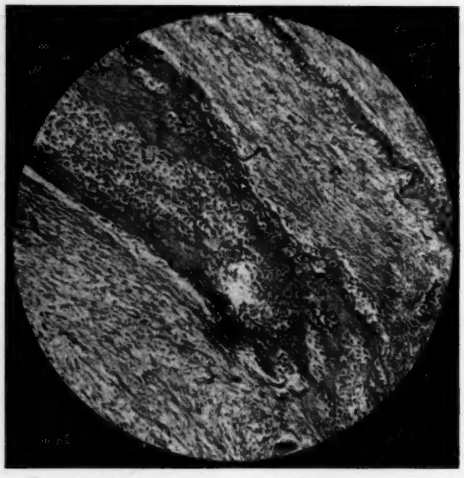

FIG. 20.—Section of the summit of the growth in the periosteal sarcoma. A radial bone spicule is in active formation by osteoblasts. Spindle-celled sarcoma alongside. (Low power.)

THE PROCESS OF INCREASE IN THIS TUMOUR AS DEDUCED FROM THESE OBSERVATIONS.—The process appears once more to be a stimulation to activity and multiplication of osteoblasts, followed later by their perversion and rapid debauch. There is clearly no essential histological, skiagraphic, or macroscopic difference between the processes of increase occurring in the osteoplastic dural and periosteal growths under examination.

A Type of Periosteal Sarcoma that proliferates on the Superficial Aspect of the Periosteum.—E. K. Martin[9] has referred to a second kind of periosteal sarcoma that, according to his description, originates from the superficial aspect of the periosteum, and, while resulting in the formation of a mass of growth in the soft tissues, has little effect on the bone concerned. *Fig.* 21 shows diagrammatically the cross-section of such a tumour of the os calcis, preserved in the Museum of University College Hospital. No new bone is seen with the naked eye, but only a little irregularity of the surface of the bone at the site of origin of the growth. Histologically the growth is a spindle-celled sarcoma with a very little irregular new bone formation in its substance near the site of attachment to the os calcis.* The tumour is invading the marrow spaces of the os calcis, and there is bony destruction and some irregular new formation. This invasion is represented in the diagram by the dotted area near the site of origin of the growth.

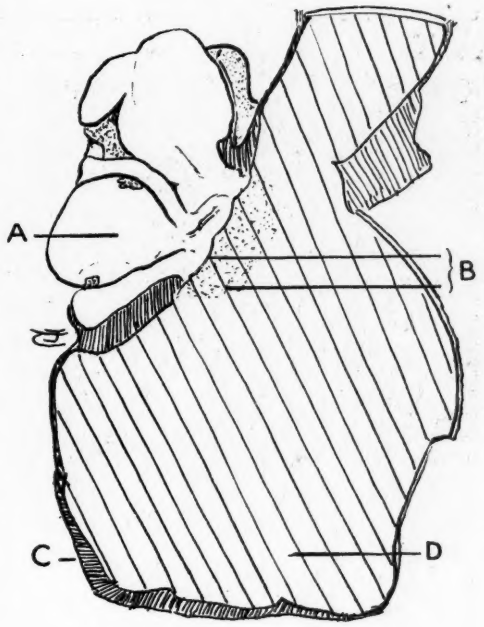

FIG. 21.—Diagram of a sarcoma of the os calcis growing from the superficial aspect of the periosteum. A, Periosteal sarcoma; B, Erosion and invasion of bone; C, Periosteum; D, Cut surface of os calcis.

COMPARISON OF THE DURAL GROWTHS WITH THE PERIOSTEAL SARCOMAS.—It has been shown that types of periosteal growth are met with that compare exactly, except in the features of rate of growth and frequency of metastasis, with the two established types of dural tumour.

An Osteoplastic Secondary Carcinoma in Bone.—This condition is well known, in particular in cases of generalized carcinoma arising primarily in the prostate.[1, 5, 10]

Skiagraphic Appearances of Osteoplastic Secondary Carcinoma.—Fig. 22 shows a skiagram of such a metastasis in the skull, for which material the

FIG. 22.—Radiogram of a slice of skull. Osteoplastic carcinoma secondary to the prostate. The radial structure is just visible in the reproduction, but easily seen in the original.

writer is indebted to Mr. F. J. F. Barrington. There is condensation of the bone of the skull, and the subperiosteal deposition of new bone in radial striæ.

* Section not reproduced.

Microscopical Appearances.—Infiltration by growth around the vessels in the vascular spaces is seen in *Fig.* 23. On the one side of the section the irregular arrangement in the calvarium itself is seen, while on the other are the radial striæ of the subperiosteal deposition. *Fig.* 24 is a higher magnification of the same section which shows the growth cells beside the vessels

FIG. 23.—Section of skull from which *Fig.* 22 was taken. Radial arrangement of bone beginning accurately at the site of the old surface of the calvarium. Growth surrounding vessels in bone spaces. (Low power.)

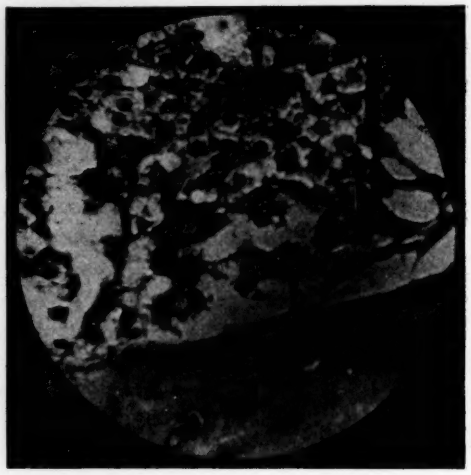

FIG. 24.—Higher magnification of *Fig.* 23. Growth in a bone space lined by bone cells.

and osteoblasts lining the bone space. The exact significance of radial striation is not under discussion at the moment, but the writer hopes to examine this aspect of the matter on another occasion.

The possible Origins of Bone in Osteoplastic Tumours of Bone.—It is obviously possible that osteoblasts that are responsible for the production of bone in such tumours may be formed as the result of proliferation of the similar cells of the bone affected. This is the origin that naturally suggests itself in the case of the carcinoma, and it is possible that in all the tumours under examination the existing osteoblasts are stimulated to tremendous multiplication and activity.

A second possibility is that the bone-forming cells are intrinsic in the tumour itself. This is, prima facie, unlikely, but not impossible, in secondary carcinoma, but is easily possible in periosteal sarcoma and the dural tumours. In this connection it is impossible to neglect the known habit of bone formation in sarcomas of the soft parts and the ossifications often seen in the intradural parts of dural tumours ([17] and elsewhere), and in the non-tumorous dura and falx.

A third possibility is that the bone formation is a tissue reaction the result of a stimulus by the tumour growth, but dependent on this stimulus rather than on the particular tissue stimulated. This possibility is supported by the known tendency of connective tissues towards ossification, e.g.,

ossification of the abdominal wall the result of chronic infections of wounds, and the ossification met with occasionally in carcinoma of lymph glands.

The writer does not presume to offer views upon the origin of the bone-forming cells, but would rather point out that in all the cases examined the microscopic characters of the newly-formed bone are identical, and that in the cases of the dural tumours and the periosteal sarcoma there is also an identity of the gross anatomy and the skiagraphic appearances. It is evident that the absence of a clear indication of the origin of the bone does not vitiate the conclusion that there is no discoverable essential difference between the bony deposits formed in these tumours and normal bony tissue.

The Habits of Spread of the Dural Tumours.—It has been shown that the histological and skiagraphic features of the spread of the dural tumours in bone are exactly paralleled by those of certain malignant tumours. It cannot from this be deduced that the dural tumours are necessarily malignant if their known habit is clinically benign (compare the melanomas). Nevertheless the behaviour of benign tumours in bone is so well known outside the skull that the contrast between them and the dural tumours is striking. In addition, the effect of benign tumours on the skull is often seen in the pituitary fossa and the internal auditory meatus, which may be largely excavated without invasion of their bony walls.

The facts of gross spread of the dural tumours are as follows: In both types a large tumour mass is found that indents but does not invade the brain. In the one type—the plaque—a large bony mass is formed by outward spread; in the other—the spherical—no such mass is formed, but the skull is invaded. In the first type the pericranium is frequently perforated, with infiltration of the temporal muscle and scalp, local recurrence in these structures being common after removal[8]. The growths are known to invade the venous sinuses, and a case has been described by Towne[11] where growth reached the left innominate vein via the inferior longitudinal and lateral sinuses and the internal jugular vein.

The benign clinical reputation thus rests upon two facts only: the brain is not invaded, and there are no secondary deposits.

To deal with the second fact first. All malignant growths do not result in remote dissemination—for example, rodent ulcer. But few rules in medicine are never broken, and rodent ulcer sometimes invades lymphatic glands.[12] The writer therefore examined the literature for records of dural tumour with secondary growth, and was at once rewarded. For example, Sir James Purves-Stewart[13] records a typical endothelioma of the falx associated with secondary growths in the lung. Towne[11] records a patient from whom was removed what was thought to be an endothelioma of the right carotid body. A year later the patient died as the result of lumbar puncture, and at the necropsy a typical endothelioma of the right side of the falx was found, that had invaded the inferior longitudinal and other sinuses. The histological picture was that of a dural tumour—spindle cells in places forming whorls. A similar description is given of the microscopical appearances of the tumour that had been thought to originate in the carotid body.

To return to the first fact—i.e., that these tumours do not invade the

brain—the antagonism between nervous and other tissues is well known and is manifested in all sorts of ways. It is shown in the rapid formation of a complete new dura where this membrane is injured, and in the slowness with which the most highly malignant glioma or gliosarcoma invades—let alone perforates—the dura. We should therefore expect that the slowly-growing malignant tumour of the dura would fail to invade the brain, however great the involvement of the superficial tissues. This is usually the case with the tumours under discussion; but again exceptions are encountered, and cases are described, notably by Oberling[14] and by Sir James Purves-Stewart,[13] where dural tumours have invaded the brain. Sir Charles Ballance[15] recorded in 1907 a remarkable case of a tumour of seven years' standing that had resulted in the occurrence of fits, headaches, incontinence of urine, and the slow development of a large frontal bony boss. At operation the greater part of the frontal lobe was found to be replaced by growth having the characteristic structure of dural tumour—that is, spindle cells sometimes arranged in whorls. Sir Charles Ballance was of the opinion that the tumour was primarily in the brain, with secondary sarcomatous involvement of the skull. He states that at autopsy the brain was not properly hardened and that it was difficult to determine the exact site of origin of the tumour. Thus, while paying the respect and attaching the importance due to the author of the record, it may be said that it is possible that this tumour having the characteristic structure of dural tumour was in reality of their number.

SUMMARY OF CONCLUSIONS.

We may regard the dural tumours as growths of slow spread, local malignancy, and occasional metastasis. Their powers of malignant infiltration are seen in their invasion of the coverings of the brain, but only in rare instances do they cross the arachnoid spaces to penetrate the brain itself. This last character of failure to cross the arachnoid they share with growths arising in the brain, which do not commonly invade the dura. They are exactly paralleled in both their main varieties by the periosteal sarcomas of the long bones, the difference between the dural and periosteal growths being found in the degree rather than the fact of malignancy in the latter.

The possible Arachnoid Origin of the Dural Tumours.—The observations given in this communication certainly in no way indicate the arachnoid as the source of origin of these tumours. Nor, be it pointed out, do they deny such a possibility, because it has been shown that malignant growths of divers characters may result in identical bony changes provided they invade the appropriate layers of the bone and periosteum.

But these findings do undoubtedly show that the normal proliferation of the arachnoid into the substance of the dura has no special and unique influence on the form of the growth. And they further suggest that the form and spread of the tumours are related to the function of the dura as a periosteum rather than as a covering for the brain.

In the presence of the exhaustive labours that have been devoted towards the determination of the tissues from which the dural tumours arise, [2, 3, 4, 7, 14, 16] the writer does not feel qualified to express his opinion

upon this subject. He rather wishes to emphasize that the facts of their form and growth are those here described ; that if these be critically examined, the dural tumours, whatever their origin, take their place naturally among the more commonplace pathological processes ; and thus the attribution to them of characters special and unique is warranted neither by clinical nor by pathological facts.

Terminology.—The writer takes no interest in terminology as such, except where words used point to unjustified conclusions or are actually misleading, from each of which vices the expression 'meningioma' is free. But he is unable to see that evidence has been brought forward to warrant the discarding of the term 'sarcoma' to denote both types of dural tumour, in consideration of their histology and habits of spread.

The writer wishes to express his thanks to Mr. Trotter and to Professor Choyce, his present and past chiefs, for unrestricted clinical and laboratory opportunites and for much informal counsel, and to Mr. Melville, radiographer to the Institute of Anatomy, University College, for the preparation of radiograms.

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THE STUMP OF THE URETER AFTER NEPHRECTOMY: THE INDICATIONS FOR PRIMARY NEPHRO-URETERECTOMY.*

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NEPHRECTOMY, first performed by Simon in 1869, remained a dangerous operation until the ureteric catheter and the tests of renal function robbed the procedure of its high mortality. Intensive study of the renal blood-vessels further diminished the risks by warding off the dangers of hæmorrhage after operations on the kidney. Researches on the perirenal fat put in the hands of urologists a method of extracapsular nephrectomy whereby improved results as regards freedom from recurrence could be achieved after nephrectomy for malignant disease, and which in cases of tuberculous kidney made it possible to obtain first-intention healing in an increasing number of such cases. The development of pyelography, ureterography, and cystography made available a complete anatomical and pathological study of both sides of the urinary tract, so that except in cases of emergency the surgeon is no longer justified in embarking on nephrectomy until he has made such investigations, not only of the side suspected of disease, but also of the side supposed to be healthy. Such studies should be designed to exhibit not only the state of the renal pelvis but also the state of the whole length of the ureter from the kidney to the bladder. It is the purpose of this paper to suggest that painstaking attention should be paid to the condition of the ureter before embarking on nephrectomy, as in selected cases the removal of the major portion of the ureter in addition to the kidney will be found to give very much better after-results than mere nephrectomy alone.

Progress in the safety of nephrectomy in the hands of professed urologists may be said to have reached its limits, but progress in the prevention of complications caused by the stump of the ureter left behind after nephrectomy has by no means as yet received the attention it deserves. More and more as time goes on the urologist will find it necessary to remove a large part, if not the whole, of the ureter at the same time as he carries out nephrectomy, and many poor results obtained in the past after nephrectomy can be traced to a failure to realize the importance of removing a sufficient portion of the ureter.

It is surprising how little attention has been paid to the fate of the lower end of the ureter left after nephrectomy. This is probably because the stump of the ureter has not given trouble sufficiently often to any one individual to make him realize the importance of the problem. When, however, any

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* A paper read before the Section of Urology, Royal Society of Medicine, on Thursday, June 23, 1927.

urologist reflects without prejudice on his past results he will probably recall that in more cases than he cares to remember the stump of the ureter has given trouble after nephrectomy, but that the trouble has either remedied itself with time or the adoption of palliative treatment, or has been glossed over. In times past when surgeons were accustomed to use silk for ligaturing the ureter it was not unusual for a stone to form round the silk ligature; and the stone was either passed with an attack of colic or had to be removed at a second operation. Even after the giving up of silk for ligature of the ureter it has happened that some weeks after an operation for nephrectomy, especially on an infected kidney, the patient has suddenly been seized with an attack of fever, renal colic on the side of the nephrectomy, and strangury. The attack has been relieved by the passage of a quantity of pus, which has included portions of the catgut ligature.

For instance, the author operated on a woman in December, 1925, and removed a foul calculous pyonephrosis on the right side, the ureter being burnt across and tied off with catgut, and the wound closed without drainage. The left kidney was healthy. On the eleventh evening the patient developed a right renal colic, followed by strangury and high fever, lasting three days. On the fourteenth evening the sudden passage of clotted pus containing the catgut ligature led to the immediate relief of all symptoms. In July, 1926, she was found to have had no further trouble, and the urine was clear and sterile.

In cases of hydronephrosis, if the obstruction to the ureter is low down in the pelvic ureter and the ureter as far as the obstruction is not removed, more often than not the lower end of the ureter left behind gives rise to trouble. The patient may complain of repeated attacks of ureteric colic similar to the attacks caused by the hydronephrosis, or the ligature may slip off and the patient be faced with a sinus continually discharging mucus or even urine, both of which conditions will call for a secondary ureterectomy. Worse still, the stump of the ureter may remain infected and cause attacks of colic, fever, and strangury. Such attacks may yield to regular lavage of the stump through a ureteric catheter. But in badly infected cases with severe obstruction at the lower end of the stump a large infected pouch may form, as Hyman[1] has shown.

For instance, the author performed an emergency operation on a woman in the country in the middle of the night in October, 1919. The patient was gravely ill, with high fever and a large tender left kidney, the right kidney being healthy. A very large infected hydronephrosis full of foul purulent urine associated with an aberrant tail artery was removed. The ureter below the artery was not dilated; yet it must have been infected. She was seen again in February, 1920. She had continued to have pain similar to her old left renal pain but not so severe, accompanied by attacks of strangury, pyuria, and fever. By means of the ureteric catheter the left ureteric stump was found to be infected but not stenosed. The right kidney was healthy. The stump was washed out several times with colloid silver through a ureteric catheter, and the symptoms cleared up completely.

The author in 1921 examined a woman 66 years of age, who since 1910 had suffered from attacks of strangury, fever, left renal pain, and profound

wasting. The left kidney was removed. It contained a stone in one of the upper calices. The kidney substance was thinned out, as was the pelvis and the whole of the ureter, which was dilated to the size of an index finger. The ureter was cut across high up in the loin and left behind. She was not seen again until March, 1927, when she stated that though she had been much better in herself, she suffered occasionally from attacks of left ureteric colic, accompanied by strangury and fever, which were relieved by the passage of purulent urine, after which the urine would remain clear for many months. The urine contained a moderate degree of pus and the right kidney was healthy. It was found impossible to pass a fine ureteric catheter up the left ureter. There existed a narrow stricture of the lower end of the left ureter, and the attacks were due to occasional retention of purulent secretion in the ureteric stump.

The contents of an infected and stenosed ureteric stump may leak into the surrounding tissues and set up a mass of dense fibro-fatty tissue containing loculated masses of pus and even urine, causing such severe symptoms as to render the life of the patient a misery unless a secondary ureterectomy is carried out, an operation that may present formidable difficulties. The cases quoted below illustrate how these complications may be prevented by primary nephro-ureterectomy, an operation which presents far less difficulty than a secondary ureterectomy, as it is carried out before the formation of these late inflammatory changes.

On rare occasions after nephrectomy for tuberculosis of the kidney the catgut ligature may slip, giving rise to a urinary fistula in the kidney wound which calls for a secondary ureterectomy. The author has met with two such cases. More frequently a pyo-ureter develops and bursts in the course of five or six weeks into the perirenal tissue and gives rise to an abscess in the nephrectomy wound. This leaves a tuberculous sinus which may refuse to heal till many months have elapsed. Such complications are to be expected if the tuberculous ureter is grossly thickened, and especially if there is definite obstruction caused by fibrosis to the exit of the contents of the infected ureter into the bladder. For these reasons Lilienthal[2] in 1911 advocated nephro-ureterectomy. Some surgeons are accustomed in all cases of tuberculosis of the ureter to remove as large a portion as possible of the lumbar and pelvic ureter; others consider that this step entails risk of the formation of an abscess or urinary fistula deep in the pelvis, and hold that it is safer to free the lumbar and pelvic ureter from the kidney and to bring it out through a small muscle-splitting incision in the inguinal region, so that it can drain on to the surface of the skin. Which is the safer method is a subject ripe for discussion. Probably all will agree that there is no need for ureterectomy in those cases where the tuberculous ureter is merely glassy with submucous tubercle and is not grossly thickened and stenosed through peri-ureteric tuberculosis.

In cases of septic stone impacted for many years in the pelvic ureter it is not always sufficient to remain content with a mere removal of the stone. If the ureteric catheter reveals that the kidney and ureter above the stone are mere dilated atrophic septic or aseptic relics, it is probably far wiser to make a clean sweep by means of primary nephro-ureterectomy.

Raymond Latcham,[3] in a paper from the Mayo Clinic, from his experimental studies on animals came to the following conclusions :—

(1) In the normal ureter after nephrectomy no attempt is made toward obliteration of the lumen by disappearance or atrophy of the mucous membrane, but there is a noticeable atrophy of the muscular coat. (2) In the hypertrophic hydro-ureter or pyo-ureter, with drainage of its contents after nephrectomy, the mucous membrane remains intact and the muscular coat gradually atrophies. (3) In the hypertrophic hydro-ureter or pyo-ureter with complete obstruction to drainage of the ureteral contents after nephrectomy, the mucous membrane remains intact and the muscular coat remains hypertrophic. (4) Absorption of the contents of a distended ureter is very much limited if it occurs at all. (5) If infection is present in the contents of the ureter, it may spread through the wall and give rise to peri-ureteral infection and abscess formation.

It seems clear, then, both from experimental and clinical evidence, that if there is no obstruction to the stump of the ureter after nephrectomy between the ligature and the bladder, the mucous membrane will not disappear, but the muscle wall will atrophy to a certain extent and ureteric peristalsis will still continue to a mild degree to extrude from time to time a small quantity of mucus secreted by the wall of the ureter. If there is a disease of the wall of the ureter—as, for instance, infection after hydronephrosis, pyonephrosis, or tuberculosis, and yet no obstruction to the lumen of the ureter—complications will seldom arise, the inflamed and infected ureter will gradually atrophy and its contents become sterile. If the process of sterilization is delayed, it can be hastened by washing out the stump of the ureter with a ureteric catheter. The author has actually observed atrophy and healing of the tuberculous ureter exposed during abdominal operations for other diseases in cases that have had nephrectomy for tuberculous kidneys some years previously, and he has noted that in many cases after removal of a calculous pyonephrosis the stump of the ureter has become aseptic and has given no further trouble. On the other hand, if there is any real degree of stricture between the site of the ligature on the ureter stump and the entrance of the ureter into the bladder, the result of congenital, inflammatory, or calculous stricture, then trouble is likely to arise in the stump of the ureter, as the muscular wall will hypertrophy in an endeavour to get rid of the contents of the ureter, causing attacks of ureteric colic ; and if the contents of the ureter are infected, the lumen of the ureter will become dilated to form an infected pouch, causing attacks of colic, fever, and pyuria, and in severe cases the infection may spread through the wall of the ureter, setting up dense fibro-fatty adhesions and even peri-ureteric abscesses.

Far greater care, then, should be exercised in taking not only pyelograms but ureterograms of all cases where nephrectomy is about to be carried out. If these show the slightest degree of stricture at any point in the pelvic ureter, then not only should nephrectomy be carried out but the stump of the ureter should be removed until a point is found above the bladder where the ureter is normal and not stenosed. If the ureter is ligated and cut off here, no trouble will arise symptomatically after the operation, and in cases of tuberculosis it is in the highest degree unlikely that any infection of the pelvic tissues or the formation of a urinary fistula will occur.

In cases that arise commonly the indications for primary nephro-ureterectomy are as follows :—

1. In hydronephrosis, simple or infected, where the stricture is low down in the pelvic ureter.
2. In pyonephrosis or atrophic hollowed septic or aseptic kidney secondary to a stone long impacted in the pelvic ureter.
3. In tuberculous pyonephrosis associated with inflammatory stricture in the pelvic ureter.
4. In all other types of pyonephrosis associated with inflammatory stricture in the pelvic ureter.

Rare conditions where primary nephro-ureterectomy is indicated are :—

1. Papilloma of the renal pelvis with secondary deposits in the ureter.
2. Primary carcinoma of the ureter.
3. Congenital opening of the ureter into the wall of the vagina associated with congenital cystic or atrophic kidney.
4. Ectopic pelvic kidney.
5. Heminephrectomy for horse-shoe kidney associated with infected stone.
6. Primary fibro-fatty ureteritis, a rare condition where infection has damaged extensively the wall of the ureter yet has left the kidney comparatively free.

SURGICAL TECHNIQUE.

The operation of primary nephro-ureterectomy can be performed in many different ways. In the majority of cases the author is accustomed to cut down on the kidney first from behind, and after the kidney has been freed and its pedicle tied, he frees the lumbar ureter from its connections, care being taken to tie all bleeding points, which may be numerous. The ureter can be burnt across with a cautery, or the kidney and ureter be left hanging out of the lower margin of the wound, which is then stitched up and the patient turned on to the back. The ureter in the easier cases where the obstruction is high up in the pelvic ureter, such as a stone, is then exposed extraperitoneally through the author's muscle-splitting incision for exposing the pelvic ureter,[4] tied off with catgut below the point of obstruction, burnt across, and the ureter with the kidney drawn out from the wound in the back. If a drain is carried to the stump of the pelvic ureter, it should consist of a piece of rubber glove : tubes endanger the walls of the iliac vessels and should not be employed. If, on the other hand, the case is a difficult one where the obstruction lies close to or even in the walls of the bladder, a better exposure can be obtained by turning the rectus muscle outwards from near the middle line below the umbilicus. Through this incision the ureter can be stripped extraperitoneally to the wall of the bladder, the numerous branches of the internal iliac and other vessels running to the wall of the pelvic ureter and bladder can be exposed and tied off, and the ureter be completely removed. In some cases it has even been found necessary to remove a portion of the bladder wall, a step which is perfectly feasible through this incision. Drainage, if considered necessary, can be carried out through a small puncture counter-opening in the lateral abdominal wall. In women

the uterine artery can be avoided by exposing the ureter in front of the broad ligament and tying it off close to the bladder, after which the ureter can be freed higher up in the pelvis behind the broad ligament, and the ureter pulled upwards and backwards through the broad ligament. The operation may be varied in certain cases by cutting down on the pelvic ureter first, and, after it has been dealt with and freed, the kidney can be exposed through a second incision and the whole specimen removed. The operation is usually more easily done in this way, but in certain cases the surgeon will probably wish to expose the kidney first and satisfy himself as to its exact condition before removing it. The operation can be performed through a long sloping incision carried from the back of the loin round the lateral abdominal wall to the edge of the rectus muscle after the method of Israels. It would appear that two separate incisions are less mutilitating and give rise to less shock, and are therefore to be preferred to this single incision.

CASE REPORTS.

The following twelve cases illustrate the range and difficulties of the operation. Only essential facts have been recorded, and no cases of tuberculosis have been included, as their treatment by means of the operation is already well recognized.

Case 1.—**Papilloma of renal pelvis, ureter, and bladder.**

A man weighing 16 stone, age 66, examined in January, 1925, for seven years had noted attacks of profuse painless hæmaturia. Cystoscopy revealed a papilliferous carcinoma on the left lateral wall of the bladder. On Jan. 22, by open operation the growth was treated with diathermy and implanted with radon seeds. No trouble was noticed until June, 1926, when an attack of hæmaturia occurred. The urine was clear and the frequency normal. At cystoscopy, carried out on June 22, the bladder itself appeared perfectly healthy, the site of the original tumour being occupied by a pale white scar.

On Dec. 3, 1926, cystoscopy was again carried out, as profuse hæmaturia had been going on for a fortnight. A papilloma could be seen peeping out of the right ureter and then disappearing out of sight as blood-stained efflux ebbed and flowed from the right ureter. A pyelogram of the right kidney showed

FIG. 25.—*Case* 1.
Papilloma of renal pelvis, ureter, and bladder.

without any doubt a papilloma in the lower portion of the renal pelvis causing a filling defect. On Dec. 8, 1926, primary nephro-uretero-cystectomy was performed. The wound healed satisfactorily. The patient shortly afterwards contracted lobar pneumonia, which proved fatal.

Specimen (*Fig.* 25).—This consists of the right kidney, ureter, abdominal portion of the vas deferens, and a portion of bladder wall surrounding the mouth of the ureter. The lower portion of the pelvis of the kidney is occupied by a villous growth or papilloma. The middle portion of the ureter is moderately dilated and contained three small blood-clots. The extreme lower portion of the ureter is widely dilated and contains three villous growths. The portion of bladder removed and the extreme lower end of the ureter are surrounded by a dense mass of fibro-fatty tissue of an inflammatory nature.

FIG. 26.—*Case* 2. Secondary carcinoma of ureter.

Case 2.—**Carcinoma of renal pelvis and ureter.**

A man, age 48, examined in October, 1918, had noted for five years hæmaturia and an occasional dull ache in the right kidney region. Blood could be seen issuing from the right ureter, and a hard nodular enlarged right kidney could be felt. The right kidney was removed. The upper end of the right ureter was blocked with a nose of papillomatous growth, the kidney very much distended and thinned, the surface of the thinned-out pelvis covered throughout with multiple papillomatous growths. Microscopic report: "Columnar-celled carcinoma of the renal pelvis". The specimen was mislaid.

There was no further trouble until May, 1920, when hæmaturia again appeared. A papilloma could be seen peeping out of the mouth of the right ureter, which was distended by the growth and its mouth prolapsed into the bladder. On May 17, 1920, under spinal anæsthesia combined with open ether, through a right paramedian incision the ureter was removed entire so as to include half an inch of the bladder wall surrounding its opening into the bladder. The latter was then stitched up and drainage established through the perineum and from the bladder itself. The patient returned to his ordinary life, but died suddenly of hæmoptysis in the following year, having had no further urinary symptoms.

Specimen (*Fig.* 26).—The ureter is thin-walled and dilated. It is partially blocked by a papilloma at its extreme lower end, and contains a small papilloma near its upper end.

Case 3.—**Primary carcinoma of ureter.**

A man, age 57, examined in October, 1919, for eighteen months had noticed intermittent but profuse hæmaturia, and since July, 1919, an aching pain in the left hip-joint. Cystoscopy revealed a smooth papilloma of malignant type peeping out of the lower end of the left ureter, which was destroyed by means of bipolar diathermy. No urine and no indigo came from the left kidney, and a ureteric catheter came up against a complete block half-way up the ureter, so that a provisional diagnosis of malignant papilloma of the renal pelvis was made.

OPERATION.—On Nov. 4, 1919, the left kidney was exposed from behind; it was found distended with blood-stained urine, the cortex thinned out, through which several hard carcinomatous nodules could be felt in the renal pelvis. The kidney was dissected out complete in its fatty capsule, the pedicle divided, and the ureter freed downwards. Four inches below the renal pelvis the ureter was found to expand and become lost in a large mass of adherent carcinomatous material. As the patient's condition seemed hazardous, and at the request of the anæsthetist, the ureter was

ligated below the kidney, burnt across, the kidney removed, and the wound closed.

SECOND OPERATION.—On Nov. 18 a second operation was performed. An incision was made one inch to the left of the middle line through the left rectus muscle, the peritoneum being brushed off the bladder and left iliac fossa and the spermatic plexus cut across. The mesentery of the sigmoid flexure was adherent to the front of the ureter. The peritoneum was then opened widely outside the sigmoid colon, which was retracted inwards. The ureter in its middle part was filled with a hard mass of growth which had pushed the common iliac artery backwards, and was adherent to and pressing on the sciatic plexus, thus accounting for the pain in the left hip-joint, and was surrounded by a dense coat of fibro-fat. The upper end was buried in inflamed fat forming a knob at least eight inches in circumference, the whole mass being adherent to the aorta for a considerable distance, which rendered the dissection difficult. The inferior mesenteric artery had to be cut across and ligatured. The upper end was freed with difficulty, but the lower end was more easily shelled out right to the wall of the bladder. The lowest inch of the ureter was not dilated—it was tied, cut across, and the mass removed. The peritoneum and the wound were then stitched up without drainage. Recovery was uneventful.

Unfortunately recurrence took place, and the patient died eighteen months after the second operation. Microscopic sections showed a primary carcinoma of the ureter. Unfortunately through an oversight all the specimens were lost.

FIG. 27.—*Case* 4. Stone in the pelvic ureter. Pyoureter and pyonephrosis.

Case 4.—**Stone in the pelvic ureter.**

A married woman, age 34, examined in April, 1927, for ten years had noticed pain in the left loin. In December, 1926, after exposure to a snow-storm, she developed a rigor, was seized with violent pain in the left side, and vomited profusely. From that day until the end of February she was confined to bed gravely ill, with fever, pain in the left loin, hæmaturia, pyuria, and wasting, but with little increased frequency in micturition.

During March her condition improved, and when seen she looked anæmic, wasted, highly nervous, and worn out by the long illness. The urine was clear to the naked eye, but contained microscopic traces of pus and blood. X rays revealed a shadow as of a stone lying in the upper portion of the left pelvic ureter. Cystoscopy showed a healthy bladder; no urine to be seen issuing from the left ureter. An opaque catheter would not pass more than three inches up the ureter and through it no urine drained away, nor could any sodium iodide solution be induced to flow up to the left kidney—a fact which, together with the absence of pyuria, suggested that the left ureter was completely blocked by a stone which was shutting up pus in the left kidney and had probably largely destroyed that organ. A skiagram showed the tip of the catheter touching the stone in the left pelvic ureter.

OPERATION.—On April 12, under ether anæsthesia, the pelvic ureter was exposed by a muscle-splitting incision in the left groin. The stone was found impacted well below the pelvic brim. The ureter, where it was normal below the stone, was

ligated and cut across. Above the stone the ureter, which was thickened, dilated, and adherent, was cleared into the lumbar region. The wound having been closed with a small drain, the patient was turned on to her face and the left kidney exposed through the back. The kidney was lobulated, thinned out, distended with pus, and densely adherent to the surrounding tissues. The kidney, ureter, and stone having been removed in one piece through the loin, the wound was closed with a drain. Convalescence was uneventful.

Specimen (*Fig.* 27).—A window has been cut in the pelvic ureter to show the stone, which has been pushed up from its original position.

Case 5.—**Stone in the pelvic ureter, calculous anuria.**

A man, age 33, first examined in December, 1924, gave the following history: In January, 1919, he had a rigor, strangury, and left renal colic. X-ray examination was negative. Urine contained pus, oxalate crystals, *Bacillus coli*. Treated by vaccines, which produced a severe attack of epididymitis on the left side. Attacks continued until August, 1924, when he entered a hospital with rigors, fever, and complete retention of urine. Suprapubic cystotomy was carried out, but nothing was found. Two months later he passed a stone naturally and the suprapubic wound healed.

On Examination the urine was found to contain pus and *B. coli*. There was pus in the prostatic fluid and in the left vesicle, and a stricture at the neck of the bladder the result of the stone. The X rays revealed a stone in the lower pole of the left kidney.

Under ether anæsthesia the stricture was dilated up rapidly. Cystoscopy showed the urine from the right kidney to be normal; from the left kidney pus and *B. coli*. Both kidneys were washed out. The patient was dieted, given urinary antiseptics by the mouth, treated with prostatic massage, bougies, and pressure irrigation of the bladder, with the result that in March, 1925, his urine was clear to the naked eye, he passed water with a good stream, the prostatic fluid appeared to be normal, his general health had improved, and he had regained his normal weight and strength.

After a year of good health, in January, 1926, he had an attack of left renal colic. Radiography showed that the stone had entered the left ureter and become impacted low down in the pelvic ureter.

On Feb. 16 he was seized with intense aching pain in the left loin, vomiting, loss of appetite, thirst, and a temperature varying between 103° and 104°. He was brought up to a nursing home on Feb. 26, when his condition was found to be one of calculous anuria, delirium, high fever, dry coated tongue; the left kidney was very much swollen, tender, and obviously full of pus. Under ether anæsthesia thick pus could be seen exuding from the left ureter like paint squeezed out of a tube.

Operation.—The left kidney was exposed from behind and removed with the upper end of the ureter. The pelvic ureter was then exposed through a muscle-splitting incision in the left groin. The stone was lying one inch above the bladder. The ureter was ligated below the stone, right against the wall of the bladder, and the whole of the rest of the ureter, distended with pus and containing the stone, was removed. Both wounds were drained. The cystoscopy and the two operations occupied one hour and ten minutes.

The ureter was grossly dilated above the stone, thick-walled, and densely adherent. There was little kidney substance left. What there was was dotted all over with tiny miliary abscesses lying between the tubules, representing a recent infection brought on by the blocking of the pelvic ureter with the stone.

The patient, stimulated with injections of caffeine and water, made 24 oz. of urine in the first twenty-four hours, 30 oz. in the second, and 40 oz. in the third. Convalescence was uneventful, and by the second week in March he was able to return to his home, his wound was healed, and he was passing 50 to 70 oz. of clear urine in each twenty-four hours.

Seen in August, 1926, his health was better than it had been for years, the urine was natural, frequency normal, stream perfect, and there was no evidence of any urinary trouble.

Case 6.—**Stone in the pelvic ureter complicating an infected congenital hydronephrosis.**

A man, age 55, first examined in May, 1927, stated that at the age of 21 he first noticed hæmaturia. On and off ever since he had experienced attacks of severe pain in the left loin and bladder, with occasional hæmaturia, increased frequency, and imperative micturition. Twelve months previously a surgeon had opened his bladder and had removed a stone, a procedure which had brought no relief.

The urine contained quantities of milky pus. The right kidney was natural and efficient. The left kidney yielded a pale, milky, purulent fluid and gave poor functional tests. The pyelogram showed a congenital hydronephrosis of the left kidney and a stone impacted in the pelvic ureter.

OPERATION.—On June 30 the left ureter was first exposed through a muscle-splitting incision in the groin, divided below the stone, and separated upwards. The left kidney was then exposed from behind and removed complete with the ureter and stone. There was a large balloon pelvis with a small lobulated thinned-out kidney riding on top of it. Recovery was uneventful.

Case 7.—**Acquired stricture of ureter. Infected and hollowed kidney.**

A man, age 24, examined in February, 1927, stated that in November, 1925, he developed a gonococcal urethritis when in Brazil. A catheter was passed and some strong corrosive sublimate solution injected into the bladder. The patient was at once seized with violent strangury and a severe spasm of pain along the course of the left ureter, and passed large quantities of blood. Ever since he had persistently passed blood in the urine in moderate amounts accompanied by pus ; he had noticed slightly increased frequency of micturition, and from time to time had suffered from violent left renal colic, followed by the passage of blood-casts of the ureter. The pain came on in spasms, was referred not only to the left loin but also to the left testicle, and was getting more severe and frequent.

The urine was smoky with blood, contained a dense haze of pus, streptococci, and blood-stained cylinders or ureteric casts. There was deep tenderness in the left loin. X-ray examination showed a large mass of calcareous glands in the left lumbar region. The prostatic fluid was healthy, and there was no urethral stricture. The mouth of the left ureter was seen to be inflamed, and as a ureteric catheter was passed to the left kidney the patient said he felt his usual pain in the left testicle. The urine drawn off contained pus and bacteria and only 0·4 per cent urea. A pyelogram showed a large hydro-ureter and a hollowed kidney on the left side. The right kidney was healthy and of good functional value ; a pyelogram of the right kidney was normal.

FIG. 28.—*Case* 7. Acquired stricture of left ureter. Infected hydro-ureter and hollowed kidney.

OPERATION.—On Feb. 16 the left kidney, cut down on from behind, was found buried in dense fibro-fatty adhesions, the ureter and pelvis much dilated and the kidney somewhat enlarged. The pedicle having been tied off, the kidney cleared

and the ureter freed into the iliac region, the wound was stitched up, leaving the kidney lying out of its lower end. A second muscle-splitting incision having been made in the left groin, and the peritoneal cavity opened, a large mass of tuberculous glands lying in the mesentery of the small intestine was removed. The peritoneum was then cut through on the back wall and the pelvic ureter exposed. As it was traced over the pelvic brim it was dilated and buried in dense iron-like adhesions, but just above the bladder it became narrowed and stenosed. Here it was cut across between ligatures, the wound stitched up with drainage, and the whole specimen removed from behind.

Convalescence was uneventful and the urine became sterile and free from pus within three weeks.

Specimen (*Fig.* 28).—The ureter is seen tied off at its junction with the bladder. Above this the ureter is dilated and tortuous and the kidney hollowed and injected.

FIG. 29.—*Case* 8. Acquired stricture of left ureter. Infected hydro-ureter and hollowed kidney.

Case 8.—**Acquired stricture of left ureter. Infected hydro-ureter and hollowed kidney.**

A married woman, age 36, examined in October, 1926, stated that in February, 1915, she got soaked when in evening dress, and was seized with fever and pain in the left side of the back. On and off ever since she has had these attacks at intervals of about a year: high fever, backache, increased frequency, and strangury. Between the attacks the bladder is usually troublesome. She has had courses of vaccines and drug treatment without relief. There was no X-ray evidence of stone. The urine drawn off by catheter was hazy with pus and the colon bacillus. The bladder, which would only hold 4 oz., showed marked basal cystitis. The right kidney proved healthy and efficient. A No. 5 ureteric catheter would not pass more than one inch up the left ureter. A fine left ureteric catheter was passed to the kidney; a specimen was taken which contained pus and *B. coli* and was functionally of poor value. A pyelogram showed the catheter curled up in a large hydro-ureter, and above this a small kidney with hollowed calices and dilated pelvis.

OPERATION.—On March 9 the left kidney was exposed from behind, the pedicle isolated and ligatured with catgut. The kidney was not enlarged or lobulated. The pelvis was not much distended, but the ureter was dilated, thin-walled, and bound down by tough adhesions, the result of inflammation. It was separated as far as the iliac crest, and the wound stitched up with the kidney hanging out of the wound.

The patient was then turned on her back, the left rectus turned outwards, the peritoneum not opened but pushed away from the left lateral wall of the pelvis, and the ureter identified. The

ureter was far larger in the pelvis than it was in the loin, tortuous, and with tough fibrous bands full of veins and blood-vessels very adherent to the peritoneum. The uterine arteries and veins, spread out fanwise across the ureter in the fibro-fatty tissue in which they were buried, had to be separated carefully and ligatured in series. With patience this was gradually accomplished, and the outer side of the ureter was cleared as far as the bladder, but the inner attachment proved more difficult to clear, and just at the lower end the peritoneum was torn. Eventually the ureter was cleared right down to the wall of the bladder. The last inch was quite narrow as it entered the bladder. Immediately above that the ureter was widely dilated and its wall thickened. It gave the impression that the large artery and veins running downwards from the uterine vessels to the side wall of the bladder and top of the vagina had compressed the lower end of the ureter and so caused the obstruction. Possibly there had been inflammation in the parametrium on the left side around these vessels which had formed constricting bands of fibrous tissue around the blood-vessels. The ureter was cut across between ligatures at the entrance to the bladder, and the whole specimen was removed entire through the loin. The wound was stitched up with drainage. Recovery was uneventful.

Specimen (*Fig.* 29).—The whole of the pelvic ureter above the ligature is seen very much dilated and its walls thickened. There is atrophy with hollowing of the infected kidney.

FIG. 30.—*Case* 9. Congenital defective kidney and ureter. Infected hydro-ureter and hydronephrosis without organic obstruction in the ureter.

Case 9.—**Congenital defective kidney and ureter. Infected hydro-ureter and hydronephrosis without an organic obstruction in the ureter.**

A girl, age 10, first examined in September, 1926. It was stated that she suffered from imperative micturition and constant bed-wetting. There was no pain and very little effect on the general health. The urine contained a quantity of pus and *B. coli*. The left kidney was natural and efficient. The right kidney was heavily infected. The right ureter was completely patulous, so that pyelographic media injected into the bladder flowed freely into the right ureter and kidney. Pyelograms showed a wide tortuous hydro-ureter and a dilated kidney pelvis with a small relic of kidney substance lying on the top of it. For some months efforts were made to disinfect the kidney and ureter by filling up the bladder with antiseptics and placing the child on a bed with her head down and feet up. The infection somewhat abated, but as the symptoms were in no way relieved it was decided to operate in June, 1927. The right kidney was exposed from behind and isolated, the ureter was then exposed through a paramedian incision, traced as far as the bladder wall, and removed in one piece with the kidney. Recovery was uneventful.

Specimen (*Fig.* 30).—This consists of a small lobulated relic of kidney substance about the size of a walnut, lying at the top of a dilated and thick-walled infected pelvis and ureter. The ureter was completely patulous at its entrance into the bladder.

This specimen represents a not uncommon type of hydronephrosis and

hydro-ureter, the causation of which is obscure. There appears to be no organic obstruction to the ureter or kidney in the lumen, in the wall, or without the wall of the ureter. The condition, which may be unilateral or bilateral, may be explained on the following hypothesis. The subject is born with a congenital defect of the ureter, just as a subject can be born with a congenital club-foot. This defect resides in the wall of the ureter, which is born so wide that the muscle coat of the ureter is from birth unable to contract so as to close the wall of the ureter in peristalsis and to close the normal valvular opening of the ureter into the bladder which exists in normal persons, but which does not exist in these defective subjects. At every act of micturition the bladder closes down in the urine in the bladder, subjecting it to a certain pressure. This pressure results not only in emptying the bladder by the urethra, but also forces urine backwards and upwards into the patulous ureter and kidney pelvis. The muscle coat of the defective kidney pelvis and ureter undergoes hypertrophy in an attempt to resist the pressure of the bladder muscle. This resistance is, however, ineffective, and gradually the kidney substance becomes hollowed out and destroyed from the back-pressure of the bladder muscle at each act of micturition. A very small increase in the pressure in the renal pelvis is sufficient when acting over many years to cause back-pressure atrophy of the renal parenchyma. For instance, in prostatics with giant overflow bladders, experience shows that on balancing the renal pressure after suprapubic cystotomy against the gravity of a column of water, this pressure is seldom more than six inches to at most a foot of water.

In this way it is possible to suggest an explanation of these cases of hydro-ureter and hydronephrosis with no organic obstruction, which are not uncommon, and resemble Hirschsprung's disease of the colon or congenital megacolon.

Case 10.—**Double kidney with double ureters, one of which opened into the bladder, the other into the vagina.**

A single woman, age 32, first examined in October, 1925, stated that in October, 1923, she had an acute attack of pyelitis of the right kidney, followed by further attacks in November, 1924, and in March and July, 1925.

The patient looked wasted and poisoned. The urine contained pus, the colon bacillus, and streptococci. Pyelograms and functional tests applied showed a healthy left kidney, but on the right side a small infected, hollowed kidney.

Operation.—The right kidney was explored from behind at an operation in December, 1925. A small narrow ureter was found running up to the renal pelvis. On the inner side of this was a thick-walled hollow structure, in appearance like a dilated ureter, which ran to the extreme upper pole of the kidney to end blindly in a small nodule of fibrous tissue. The renal artery and vein, which coursed in front of the double ureters, were ligatured with catgut, the kidney was pulled downwards and outwards, and the lower portions of the double ureter were defined as far as the iliac crest. Here the two ureters were divided between ligatures, and the wound was stitched up without drainage.

On investigating the specimen removed (*Fig.* 31) it was found that the lower outer ureter ran to a moderately dilated renal pelvis, as shown in the pyelogram, which pelvis belonged to the lower half of a double kidney. The upper inner ureter, dilated and thick-walled, ran to the extreme upper portion of the kidney, where it ended in a small nodule of tough fibrocystic tissue. This tissue was found to be an atrophied bud representing the upper half of a double kidney.

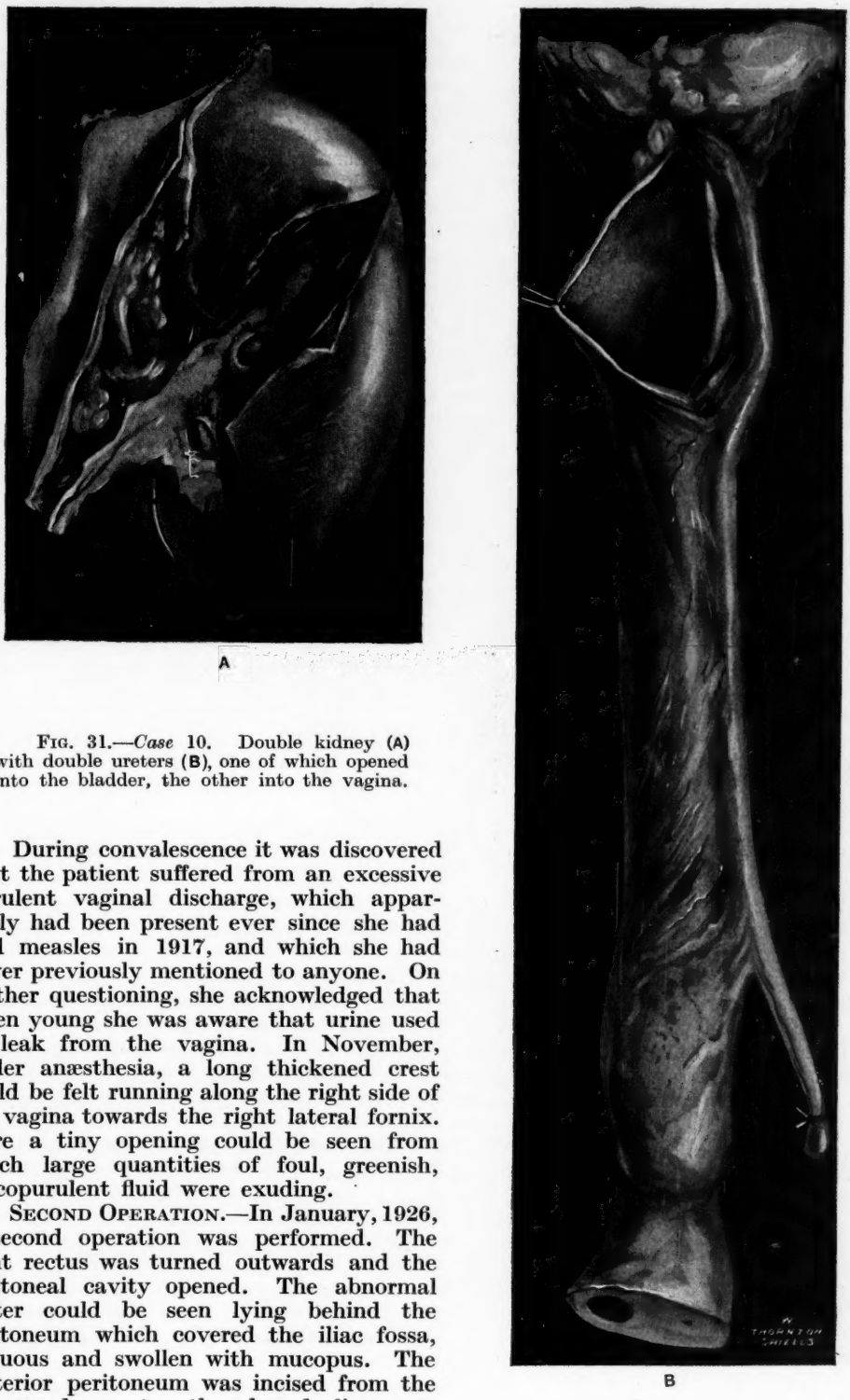

FIG. 31.—*Case* 10. Double kidney (A) with double ureters (B), one of which opened into the bladder, the other into the vagina.

During convalescence it was discovered that the patient suffered from an excessive purulent vaginal discharge, which apparently had been present ever since she had had measles in 1917, and which she had never previously mentioned to anyone. On further questioning, she acknowledged that when young she was aware that urine used to leak from the vagina. In November, under anæsthesia, a long thickened crest could be felt running along the right side of the vagina towards the right lateral fornix. Here a tiny opening could be seen from which large quantities of foul, greenish, mucopurulent fluid were exuding.

SECOND OPERATION.—In January, 1926, a second operation was performed. The right rectus was turned outwards and the peritoneal cavity opened. The abnormal ureter could be seen lying behind the peritoneum which covered the iliac fossa, tortuous and swollen with mucopus. The posterior peritoneum was incised from the cæcum down to the broad ligament,

exposing the double ureters, which were deeply buried in dense fibro-fatty adhesions full of veins. The upper ends ligatured at the first operation, buried in a mass of retroperitoneal fat, were freed and brought downwards and forwards into the pelvis until the uterine vessels were reached, crossing from without inwards and over the ureter, and were cut across between ligatures. The small outer ureter was found to pass across the front of the upper dilated ureter and run forwards through the broad ligament to the back of the bladder. This was separated and cut across between ligatures. The dilated upper ureter was traced outwards to the top of the right lateral vaginal wall, where it was surrounded with dense fibrous bands enclosing veins and arteries, which made dissection very difficult. At this point it divided into two branches. The inner one communicated through a fine opening into the right lateral fornix with the vagina itself. The outer one ran downwards and outwards for some inches as a blind pouch along the lateral wall of the vagina, corresponding to the ridge previously noticed in the vaginal wall. A clamp was then placed as low down as possible, and the two branches of the ureter were cut across below the clamp. When this was done a certain amount of purulent fluid escaped from the blind pouch still attached to the vagina. This cut left a wide hole leading from the upper end of the blind pouch along the lateral wall of the vagina into the upper end of the vagina itself. A tube was inserted through the hole into the vagina and left to drain the space opened up in the retroperitoneal tissues. The cut in the posterior peritoneum having been stitched up and the appendix removed, the wound in the abdominal wall was stitched up as usual. The wound healed satisfactorily, but the patient continued to suffer from occasional attacks of dull pain in the pelvis, always reduced by a sudden discharge of pus from the vagina.

At a further examination under anæsthesia in May, 1926, a small cystic swelling could be felt on the right side of the uterus which opened into the right side of the fornix and was continuous with the thickened crest which ran from the opening downwards and outwards along the right wall of the vagina. This crest and as much as possible of what remained of the blind pouch were cut away through the vaginal wall with scissors.

Since then the patient has gone on well, and has been able to hunt and live an active life, although she occasionally gets mild attacks of pelvic pain relieved by the passage of a small quantity of mucopus from the vagina.

Specimens (*Fig.* 31).—

A. Consists of a double kidney with two ureters. The upper kidney is represented by a small fibrous nodule lying at the top of a wide, thick-walled ureter, heavily infected.

B. This consists of the lower portion of the two ureters. The small lower ureter crosses the upper ureter in front from without inwards and ends in a ligature at the point where it entered the bladder.

The upper ureter is widely dilated and the wall thickened by inflammation. It runs downwards and outwards and is seen to end in two branches: the inner one is small and terminates by an opening into the top of the vagina, the outer one is larger and was continued in life some three inches farther down to end as a blind pouch lying in a thick-walled crest in the right lateral wall of the vagina. This pouch, not shown in the drawing, was removed at the third operation.

The full diagnosis of this case was only possible at operation, especially as the patient had concealed a most important part of her history till after the first operation.

Case 11.—**Congenital cystic kidney with ureter opening into the vagina.**

A child of 12 years, first examined in 1913. It was stated that since birth her mother had noticed large quantities of pale urine leaking continuously from the vagina. An opening could be seen in the left lateral fornix of the vagina, from which pale urine containing 0·2 per cent urea exuded,

Operation.—The left rectus was turned outwards. A small cystic kidney was found on the left side, and the ureter could be traced down from this to the top of

the vagina. The kidney was freed from its pedicle, and the ureter cleared to the top of the vagina and cut across. The kidney and ureter were then removed in one piece. Convalescence was uneventful, and there was no further trouble.

Case 12.—**Congenital ectopic pelvic kidney complicated with calculus and infection.**

A woman, age 44, examined in April, 1927, stated that for sixteen years she had suffered from intermittent attacks of fever, bladder strangury, and pyuria. Recently she had suffered from severe attacks of pain in the front and back of the left loin, with nausea and faintness. X-ray examination revealed a curious group of shadows shaped like gall-stones lying in the middle of the pelvis. Pyelography revealed a healthy right kidney lying in the normal position, and an infected ectopic hydronephrotic left kidney of poor functional value lying in the pelvis and containing stones.

OPERATION.—In May, 1927, the left rectus was turned outwards from the middle line, the peritoneum opened, and the intestines cleared out of the pelvis. The left

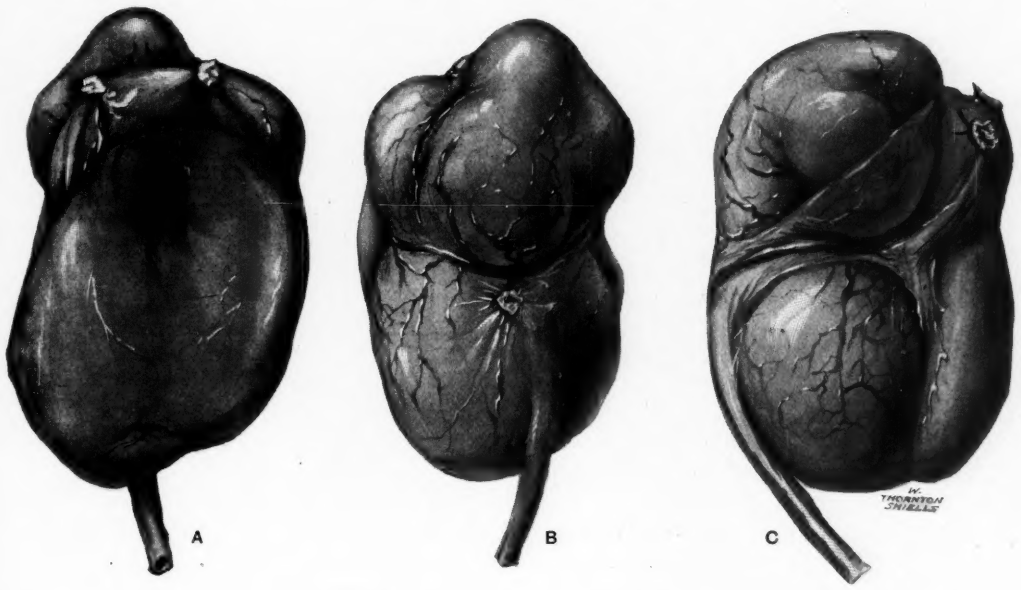

FIG. 32.—*Case* 12. Congenital ectopic pelvic kidney complicated with calculus and infection. A, Front view; B, Back view; C, Side view.

kidney was lying behind the peritoneum below the brim of the pelvis. The kidney itself lay on the left side of the dilated pelvis, from which the ureter passed downwards and backwards behind the kidney to penetrate the left broad ligament and reach the bladder. The kidney was supplied by three sets of blood-vessels. There were two arteries and two large veins running down from the front of the aorta and vena cava over the bifurcation of the aorta to enter the upper end of the kidney and suspend it. Large veins emerged from the main veins to distribute themselves in the perinephric fat. These had to be tied in series, the peritoneum being cut through and the kidney and pelvis gradually cleared. The two upper sets of blood-vessels were ligatured with catgut and the vascular pedicle was freed. The ureter was then traced down through the broad ligament to the wall of the bladder. There were two or three large arteries and veins running up in a plexus from the uterine artery and vein, surrounding the ureter and passing up to the kidney substance. These had to be ligatured in turn, and finally the ureter was left attached to the wall of the bladder free from blood-vessels. The ureter was tied off close

to the bladder wall and the whole specimen removed. The peritoneum having been stitched up on the back wall with catgut, the abdominal wound was united without drainage in the usual manner. Recovery was uneventful.

Specimen (*Fig.* 32).—

A. The kidney is viewed from the front exactly as it lay in the body. The kidney substance is below and to the left. Above there can be seen the ligatures round the two upper groups of blood-vessels, between and below which is a large venous sinus. Above the ligature can be seen a portion of the dilated kidney pelvis.

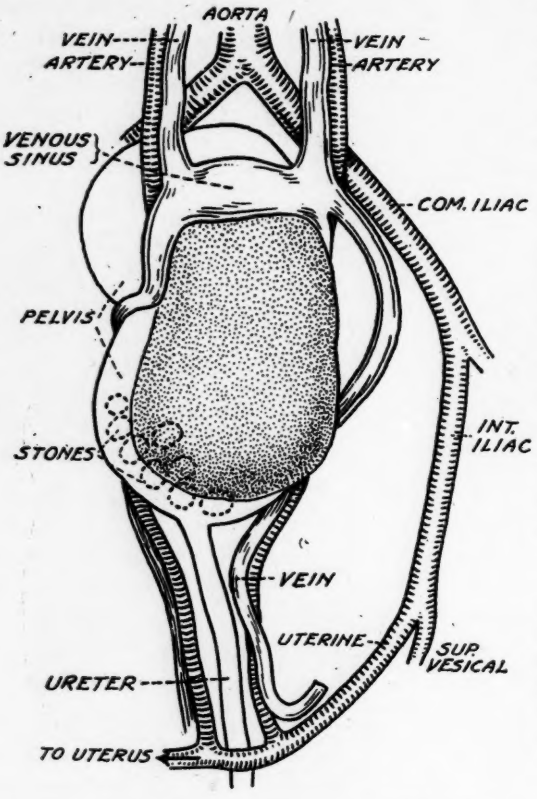

FIG. 33.—*Case* 12. Diagram to show the blood-supply of the kidney in *Fig.* 32.

B. The kidney viewed from behind and hidden by the grossly distended renal pelvis. The pelvis is constricted around its middle by a wide band of fibrous tissue containing a large vein which forms another venous sinus connecting the lateral veins on each side. The ureter comes off underneath this band and vein, and is constricted by them so as to possess a valvular opening.

C. This shows a lateral view of the specimen. The kidney is in front, the dilated pelvis behind. The origin and constriction of the ureter by the band and vein are made clear.

Fig. 33 represents the main blood-supply of the organ from three different sources, and indicates the stones lying loose in the renal pelvis. In addition to the main blood-vessels and their anastomoses as shown, there was a set of six large anastomosing veins running in front of the specimen in the perinephric fat which had to be tied off and removed before it was possible to expose the kidney itself.

The extraordinary wealth of the blood-supply of the pelvic kidney is a feature met with in pelvic ectopic kidneys, and has been known to deter operators from proceeding to nephrectomy in such cases. The exercise of gentleness and patience is required to surmount this difficulty.

Though the kidney was lying in the pelvis, yet the renal pain experienced by the patient was in the left loin in the usual position of renal pain. The writer knows of three similar cases operated on by others in which the same feature as regards the position of the pain was noticed. This fact makes the diagnosis all the more difficult.

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THE EARLY DEVELOPMENT OF HOSPITALS (BEFORE 1348.)

(Being the Thomas Vicary Lecture delivered at the Royal College of Surgeons of England, on November 3, 1927.)

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I DESIRE in the first place to express my sense of the honour of being invited to address the Royal College, and also my pleasure at seeing here to-day representatives of the Barbers' Company who endowed this lecture. It is a striking thing to reflect that 500 years ago they were taking their share in founding surgical lectures—just as they are doing to-day.

In the twelfth and thirteenth centuries of our era, a remarkable enthusiasm for creating hospitals came to a head. It had grown up gradually, and spread over many distant countries, from Burma, Siam, and Ceylon, to Syria, Persia, Egypt, and the whole of Western Europe, excepting perhaps Russia. I propose to inquire what were the sources of this movement, and how it was that hospitals had grown up everywhere in that huge area.

Now an institution or idea may arise in the progress of civilization in two or more distant places simultaneously, and there has been much discussion whether such things really have a common origin, or start independently. Thus, did the institution known as a hospital start from a single source, and the idea of its utility spring from a single mind? The earliest beginnings we know of can be traced to about the sixth century B.C., in places far apart both in the West and distant East, and hospitals do not appear in earlier times or outside a certain group of countries, with the one exception of Mexico. Thus there is no evidence of them in Assyria, Babylon, early Egypt, or China, in spite of the medical skill and civilization there existing.

The idea once started, however, appealed to various religions and to various rulers of men. The Buddhist or Christian ascetic, who neglected his own body, still toiled to build hospitals for the comfort of the sick. Among founders were many blood-stained Indian, Roman, or Moslem princes, Greek city oligarchies or tyrants, half-savage barons, the Kaiser Karl the Great, Haroun Ar Raschid, and our own Athelstan. In Western Europe, especially in the twelfth and thirteenth centuries, we find a host of private persons and societies competing with their rulers in the work. Two or three records will give some idea of the results at that date. Thus in France in 1226 Louis VIII left legacies to 2000 hospitals and to 200 poor-houses. In England, besides monastic infirmaries, 577 hospitals and asylums were founded between 1100 and 1400. In Germany and Italy the numbers were huge; one society alone is credited with building 900 in the thirteenth century. In Ceylon and Burma they seem to have been ubiquitous, and probably, too, in the shrunken Byzantine Empire. In the great Moslem world at least twenty cities boasted of

having one or several hospitals each, some of them being the most perfect of that age. The enthusiasm only faded about the time when the Black Death and the invasions of the Mongols and other barbarians wiped out a large part of the builders and the patients. We may add that it was revived suddenly, at least in Western Europe, in the eighteenth and nineteenth centuries, but in the history of the idea such ebbs and flows had occurred before.

Let us pause to consider what is meant by a hospital. In the strict sense it is a place designed for the residence under favourable conditions of the sick or wounded during treatment, or during the progress of a self-limited disease. The better class of hospitals, too, provides for the observation and registration of symptoms, for baths, surgical theatres and instruments, laboratories, and other facilities for treatment. Many, too, provide for the training of students, surgeons, and physicians.

We must distinguish them from (1) rest-houses for travellers, (2) orphanages, and, (3) homes for the poor and aged. On the other hand, asylums for the insane or for lepers, or consumptive sanatoria, may be either true hospitals or merely homes for segregating affected persons, in fact quarantine stations. Again, out-patient dispensaries present some difficulties, since elaborate treatment, as in many eye clinics to-day, researches, and the training of students may be carried on in them; but, if we are to regard them as hospitals, how about the private office or surgery of a practitioner? They are all institutions for the cure of disease, and the question of ownership is unimportant. However, ordinary usage tends to restrict the term hospital to places with beds for the patients, and I think quite rightly. Many ancient institutions combined true hospitals with other departments under the same roof, such as orphanages and asylums for the poor and insane. The mediæval hospital was usually like our Poor Law infirmaries, where both acute and chronic cases are taken in, nursed, and treated. It is not always possible to say what kind of institution historical records refer to, though Justinian carefully separates nosocomia from poor-houses, rest-houses, orphanages, and homes for aged persons or foundlings. I shall endeavour to restrict my remarks to what were true hospitals wholly or in one department, though a hard-and-fast line is impossible. It is astonishing how perfect in every detail some of the very ancient ones were, but as to others we know nothing but a single word in a charter or inscription.

Let us now take up the story of the rise of hospitals in the various countries.

THE BUDDHIST HOSPITALS.

It is not clear to me that any sprang up in the life of Gautama; but after his death in 543 B.C. monasteries very quickly arose throughout the Buddhist world, each containing an infirmary open to any sick man, or, indeed, to animals. These hospitals, formed to carry out the precepts of the religion, existed then in great numbers from 500 or 543 B.C. We have no evidence that the idea was taken from any previous institutions, either Indian, Persian under the first Achæmenians, or from an early Indo-Mediterranean world. They were clearly prior to the Grecian influences of Alexander's invasion, and at present we can only regard them as a new idea. In the *Arthaśastra*,

book ii, cap. iv, possibly written by Kautilya under Chandragupta about 325 B.C., we find hospitals spoken of as a necessary thing in fortified towns. These would probably be military hospitals, as distinct from the religious foundations. In the second Edict of Asoka about 257 B.C. we read :[1] "Everywhere in the dominions of his Sacred and Gracious Majesty, and among his frontagers as far as Antiochos [theos] the Greek king, and the neighbours of that Greek king, have healing establishments for men and animals been set up and medical herbs have been imported or planted." Thus he claimed that not only in India, but also in Syria and Mesopotamia, had such Buddhist institutions been founded. However, no trace of them has since been discovered in those western lands. Let me recall to you a few of the other well-known royal foundations. King Gamani in Ceylon, B.C. 161, claimed that he had maintained hospitals in 18 places. King Buddhadasa in 341 A.D. not only provided hospitals for men and animals, but practised surgery himself.[2] Fahien, the Chinese pilgrim, 405–11 A.D., describes the excellent free hospital supported by benevolent chieftains at Pataliputra ;[3] all the sick from the provinces near came there, were attended by a physician, but stayed only so long as they were ill. King Harsha, after 30 years of warfare, about 643 A.D., established hospitals throughout his empire in imitation of Asoka. Huen Tsien, another Chinese, tells us that these 'houses of benevolence' existed in the Punjab, in Central India, and in two other provinces. Recent opinion places in this Buddhist period the really able surgeons Charaka and Susruta, men who, as is well known, practised laparotomy, suture of the intestines, and rhinoplasty. Still later on, in 1190, Parakrama in Ceylon is found hospital building, one being mentioned of great size and splendour.

Thus from before 500 B.C. downwards Buddhist hospitals were perpetually being built (1) by victorious rulers, (2) by monasteries, or (3) by individuals. Where Buddhism died out, as in India proper, they vanished, except at Surat and a few other places, but they still exist to-day in Burma, Siam, and Ceylon.

ZOROASTRIAN HOSPITALS.

Of Zoroastrian early hospitals very little is known, though we might expect to find them under that religion. It has been said, indeed, that a great part of the Persian records were destroyed by two conquerors, Alexander when drunken, and the teetotal Arabs under Omar. However, the *Avesta* and a great deal more survived, but there is so little recorded on the subject, that it has been thought that Persian medicine was really negligible, and in support of this it is pointed out the kings were so keen on getting Greek doctors for their courts that they kidnapped them when they could not hire them. Still, in the *Dinkard*, which seems to be an abstract of earlier liturgies, it is laid down as one of the duties of kings to found hospitals in important centres and to provide physicians and drugs.[4] We do not know, indeed, how far the kings in the humane and well-organized Persian Empire carried out this rule until we come to the important embassy sent by Chosroes I or Khusraw to India about 550 A.D. to study the Buddhist medical system, and to bring back drugs to Gondishapor. Here was a flourishing medical school composed partly of Zoroastrians and partly of Nestorian Christians, and soon a great hospital

appears there also. Dr. Bargoia's mission to India found the Buddhist hospitals in great activity, and he was able to bring over teachers, drugs, and many things which greatly impressed his contemporaries. Thus, it is claimed that the game of chess, and certain popular Indian tales, the Fables of Pilpay, were brought by him, and, curiously enough, among the drugs which were first introduced there into the Western world about that time was cane sugar.

This hospital and school of Gondishapor combined then Buddhist, Greek, and Zoroastrian teachings, and it flourished vigorously. Its influence spread far and wide, and in later times notably among the Moslems. For many generations it was the chief source of learning in the East, as Clifford Allbutt remarks.

HOSPITALS IN THE WESTERN WORLD.

These originally started from two sources, the Æsculapian cult on the one hand, and on the other a provision for sick citizens devised by the practical Greeks, who were intent on forming a model city state, and had a large body of able medical men at their command.

1. *The Temples of Asklepios*, beginning perhaps with Epidauros about 500 B.C., claimed many cures, which in early times were said to be produced by the direct action of the Numen, but in later days generally through remedies prescribed by the god to the patient while sleeping in the shrine. Hence the term incubation. The remedies included baths, ointments, bleeding, massage, operations, and drugs. Fees were certainly charged to the well-to-do, and apparently beds were provided for patients while the remedies suggested were being applied. It is estimated that there were over 320 of these temples, and of these nearly 200 existed before the time of Alexander the Great. There were also similar healing shrines of Pluto, Amphiarios, Dionysos, and, from the time of the first Ptolemy, others of Isis and Serapis, the latter being a somewhat doubtful divinity who took the place of older Egyptian gods such as Ptah and Imotep. The relation of Greek and Egyptian incubation shrines is most obscure, but I know of no evidence showing that the Egyptian were earlier. To show how widely the incubation cult had spread, we may mention that Arrian speaks of seven of the companions of Alexander as sleeping in the temple of Serapis at Babylon to inquire as to their leader's illness. He has been criticized for this statement, but it seems that the temple was really one of Baal Zipur, who was later on identified with Serapis.[5]

Now Cos was originally an Asklepian shrine, and in later times it possessed a magnificent hydropathic establishment or hospital, the ruins of which still exist. Possibly this hospital was of a different origin, and due to the great medical school which had grown up there from the time of Hippocrates; and this brings us to the second class.

2. *Public Health Officers.*—Side by side with the temple shrines were great numbers of private secular doctors carrying on their art, and also a highly-paid and well-trained public medical service even as early as 600 B.C. Democles, for instance, a little later, about 500 B.C., is found drawing a salary equal to £250 and £500 per annum as city physician. These public medical officers appear not only in all cities of Greece itself in the fifth or sixth century, but also in Sicily and Southern Italy, and can be traced from the time of Plato

down to Roman times everywhere. Hence we can, I think, very well accept the conclusion reached by our late friend and teacher, Clifford Allbutt, that there was public provision universally for the sick citizen, first in Greek cities and later on in the Roman Empire.[6] Now there were not only physicians in all Greek cities. There were hospitals as well, for all doctors, public and private, had *iatreia* or dispensaries, and in some of these were beds for in-patients from about the fourth century downwards. Many of the public dispensaries were large buildings with a numerous staff of attendants.

In the Roman City and Empire, too, some time before our era, there were clearly great and well-equipped hospitals, *valetudinaria*, for well-to-do persons as well as for the poor and slaves. The staff attached to one of these hospitals included not only medical men but also assistants, dressers, and librarians or registrars. It is curious to find Celsus speaking of doctors at large hospitals having so many patients that they can hardly give them the individual care they need. On the building, cleansing, decoration, and cookery thought best in these *valetudinaria*, we have actual treatises by Columella, Vitruvius, and others. As to military hospitals, though there is plenty of evidence of a medical service in Greek armies and possibly in Persian ones, there is no mention of hospitals; but in Rome from the time of Augustus the military hospitals were excellent and well arranged. Thus Haberling and Allbutt speak warmly of the one at Carmuntium, 25 miles from Vienna, in the first century, and of Novesium on the Rhine under Tiberius and Claudius, built on the corridor plan with many small wards. Under Hadrian we read of others.

Thus, in the pagan world, hospitals were founded (1) at sacred shrines, or (2) by municipal care, or (3) by private practitioners, or (4) by military rulers.

There is reason to think that between 100 and 300 A.D., in the social troubles of the Roman world, municipal and possibly other hospitals fell largely into decay. Then Allbutt states definitely that Constantine in 335 A.D. decreed that all public hospitals should be closed and their staffs dismissed, but later on he allowed some to be reopened. This reminds us of our Henry VIII and the London hospitals. One would like to be clear whether it was not solely directed against pagan shrines, if such a decree was issued, but I confess that I can find no evidence for the statement.

CHRISTIAN HOSPITALS.

In the Roman world great changes and a remarkable expansion of the hospital system gradually took place under Christianity.

1. Municipal hospitals are little heard of for a very long period, but the official hospitals of the bishops become important.

2. Military ones under Byzantine rulers are said by Bury to have been good and well equipped; otherwise we hardly hear of them till the time of the Crusades.

3. The emperors and kings founded a fair number of civil hospitals.

4. The sacred shrines had successors in some Christian churches.

5. Though the *iatreia* of some private doctors long survived, medicine and the other arts died out almost entirely in large areas.

6. Individuals and voluntary subscribers founded vast numbers of hospitals wherever the barbarian invaders and the wars allowed.

7. After 529 A.D. monasteries took up the work in Italy, and later on in the northern countries.

The Bishops' Hospitals.—In the first age of Christianity the house of every Christian might be used as an infirmary, as Pelliccia says,[7] but from the time of Constantine or earlier it was customary for each bishop to set up a hospice with beds for the sick close to his own house and cathedral.[8] This is repeatedly mentioned. Thus the fourth council of Carthage, 436, made it compulsory for that region, the hospice to be *non longe ab ecclesia*. Sometimes the bishop managed it personally: thus we find Augustine taking his meals among the sick; usually he appointed one of his canons to do so. Later on in Rome a regionary deacon, and still later a monk, was put in charge. The council of Chalcedon insisted that the manager of a hospital must be under the control of the local bishop. Like episcopal schools they were supposed to exist in every diocese, but their success was variable. In some places the bishop was too poor to provide more than a rest-house; in others a good hospital appeared, and even a school of medicine.

Charlemagne in his capitularies of 803, and the Councils of Aix la Chapelle 817 and 836, lay down that a hospital must be attached to each cathedral and even to each collegiate church and monastery. At Orleans in 549 it was enacted that the bishop must maintain the hospital which Childebert had founded. At Lyons in 583 the bishop of each see is required to provide for and segregate the lepers. Notable instances of episcopal hospitals are seen at Cæsarea under Basil in 370, Merida in Spain 580, where the great revenues and the physicians and nurses are mentioned, as well as the fact that Christians or Jews, slaves or freemen, were all taken in and treated. Then we have Chrysostom's foundations at Constantinople, one at Ephesus, another at Hippo, and in a later age the Hôtel Dieu at Paris long managed by the Chapter of Notre Dame, those at Chalons, Tours, Rheims, Cologne, Bremen, Constance; and in England at Worcester, Lincoln, Canterbury, and Winchester. As we shall see later on, the management was finally handed over to the lay magistrates and revived municipalities in many countries.

Under Justinian startling legislation took place as to the bishops in other matters. Roman law had previously provided very little protection for charities against fraud and peculation. Justinian now took the bishops and made them guardians and public trustees of all charities, hospitals included. Thus, if a legacy appeared in a will to found a charity, it became the bishop's duty to appoint an officer to enforce the proper performance of the legacy, and they received power to visit all institutions and to correct abuses.[9] All this is quite distinct from their duty to found and manage their own diocesan hospital. In the breakdown of the State this visitorial power became the only protection charities had, but it was a great tax on the time of the bishops and their councils. We find it lasting all through the Middle Ages and asserted in the Councils of Vienne, 1311, and Trent, 1545. Thus the Council of Durham, 1217, said, "Those who wish to found a hospital must receive from us rules and regulations", much as they would apply to-day to the visiting justices.

Imperial, Royal, and Papal Foundations.—Of these may be noted especially those at Rome by Symmachus in 500, by Belesarius in 550, and by Pelagius II in 560, at Lyons by Childebert about 500, at Autun by Brunehaut in 595, at Jerusalem by Justinian about 510, and again at Constantinople, where he gave up one of his palaces to form a new one. That town obtained another from Isaac II in 1190. Alexius had already in 1116 rebuilt and enlarged a huge infirmary there, said to shelter 10,000 inmates. Our king Athelstan had endowed or founded St. Leonard's at York in 937. This had afterwards 220 beds as well as an orphanage under the same roof.

Christian Shrines.—After the coming of Christianity the populace long adhered to the Asclepian shrine treatment and worship. To meet the difficulty various churches were built, and the people were induced to resort there to implore the intercession of the saint or martyr commemorated in them, the theory being that the One God the Healer and Saviour of all men might grant a cure in response to the prayer of his servant the saint or martyr. Crowds of sick came to pray, and many claimed to be cured. Even incubation in its old form soon appeared, and the kindly saint was believed to give advice in visions to those who begged for his intercessions with the Almighty. Here and there followed hospices for the waiting sick in which baths and medical treatment were given. Thus the churches named after Cosmas and Damian replaced the shrines of Castor and Pollux, Cyrus and John drew off the worshippers of Isis or Manuthes, and the prayers of St. Michael were sought at Sosthinium in place of the cures of Apollo or Serapis. This cult has survived to the present day, with hospices at Cyzicus, Daulia in Parnassos, and with incubation at Tenos, to mention only a few.[5]

Monastic Foundations.—We need not spend much time over the well-known and great work done by the early orders. St. Benedict gave a very high place to the care of the sick. *Infirmorum cura ante omnia et super omnia adhibenda sit*, says his rule. Cassiodorus urged the need of medical study and collected a little library of medical books for the brethren. The infirmaries for the monks, and in some places for outsiders, were built with extraordinary care, and regardless of expense; they began in Italy from 529 and spread everywhere there, but not in France, Germany, or England until about 800, and during the utter breakdown of civilization the monks almost monopolized medicine. It has been said that Benedictine houses reached 37,000 in number. Whether that is true or not, in the Cluniac branch every house was ordered to build an infirmary for monks and a hospital for outside patients. Of course there was always a minority, like the Christian Scientists and others to-day, who were opposed to medical treatment. These relied on prayers and charms, but that does not affect the enormous sums which the majority spent on hospitals for the sick, and such medicines as they had. In England, the monks of St. Alban's built their infirmary in 794, and it was followed by a host of others. In many places, where no other doctors existed, there was a constant drain on a convent to send out skilled monks to attend the sick.

Individual Founders.—Besides the official work of the Christian society, it was the first duty and privilege of each individual to care for the sick and poor. Compassion for the sufferers, says W. H. S. Jones, was a virtue amongst Pagans: it was *kalon*; but amongst Christians it was *deon*, a duty. Hence

occurred a huge new outburst of hospitals founded by individuals or by voluntary subscribers as soon as it could be safely done. For example, Zodicus, about 330, collected the lepers in Constantinople into a hospital, the wealthy Olympia and others helping in the work. Hospitals there soon multiplied so fast that Ducange has reckoned 34 hospitals and asylums in it, but many of these were gifts from kings and bishops. At Edessa, in 372, St. Ephrem collected subscribers for a hospital of 300 beds, which was refounded in 410 by Bishop Rabbouli with another for women. Here a brilliant medical school grew up which carried Greek medicine to the Far East. At Rome, Fabiola in 380 gave her wealth to a new hospital in which she worked as a nurse. St. Paula did the same near Jerusalem. We find St. John, the almsgiver, building seven hospitals for women at Alexandria.

To take another instance from the charming history of Sorore of Sienna about 898, the William Law of his time. How he began with one poor wretch lying in the street, and gradually built the great hospital of St. Maria del Scala, and got together subscribers, a staff, and a lay committee, all so well organized that ten other Italian cities came to get him to introduce his system and trained helpers there also.[10] Lastly, as in private duty bound, I pray you to remember our own Rahere founding St. Bartholomew's in 1123 with help from rich and poor alike. These are only a few instances of the munificence of the war-stricken and dismembered Christian world.

MOSLEM HOSPITALS.

These mark an important stage in our story, and since the empire of the early Caliphs soon became highly civilized and wealthy, hospitals and the medical art made extraordinary progress. The relief of the sick and poor was also a duty in Islam, and the new state drew its inspiration as to medicine and hospitals from two sources. First, there were its Byzantine neighbours, who had countless hospitals. Then, besides this, Nestorian missionaries, doctors, and entire medical schools had been exiled to Asia. It has been said that Syrians and Egyptians, being strong home-rulers, took up any heresy condemned by Constantinople, and when persecuted went over the border with their Greek literature and science, or in later times often supported the Moslem invaders. The Persian kings gave some toleration to the earlier fugitives, and their medical skill won them favour. One group settled at Gondishapor and joined the staff of the great school and hospital there. As we have seen, Chosroes by his embassy to India obtained for it the teaching and remedies of the then flourishing Buddhist hospitals. This school, derived from Greek, Persian, and Indian origins, was the second source of Moslem knowledge. Even the Pagans of Harran were propagators of Greek medicine and translators of texts. Al Harith, one of the alumni of Gondishapor, attended Mahomet himself.

The first hospital of the Caliphs was one set up at Damascus in 707 A.D., by Al Walid, for eye cases, for lepers, and for the ordinary sick. His cousin Khalid had studied chemistry under an Alexandrian teacher, and we may ascribe this hospital to Byzantine influence. The invaders of Spain quickly founded a hospital at Cordova, and in Egypt, in 873, Tulun built one at Cairo,

and used to go round the wards himself to inspect them, till one day a mentally deranged patient threw a ripe pomegranate at him and spoilt his robes.

Turning back to the other source, we find the Caliph Al Mansur, in 765, sending for George Bukt Yishu, the chief physician at Gondishapor, to attend him.[11] Other physicians and also translators of Greek medicine followed, and hospitals sprang up so fast that about 790 Haroun Ar Raschid actually ordered that a new hospital should be erected for each fresh mosque in Baghdad, curiously echoing the words of his distant friend Charlemagne. Again, in 840, we find the mother of the then Caliph is founding another, and in Persia, now a Moslem country, a great hospital appears at Rhay or Rhazes near Teheran. Here we learn that Abu Bakr, afterwards so famous in Europe as Rhazes the physician, was chief of the staff with pupils and clinical clerks. He, too, was sent for to Baghdad to preside over the great Jahya hospital about 905. Two fresh ones were also erected, and the Jahya itself was rebuilt with great magnificence by the Caliph Azudu'd Daula in 976, and then was known as the New Hospital near the bridge on the west of the city.[12] It had a staff of 24 physicians, and special wards for fevers, eye cases, accidents, and various other diseases. The Caliph also determined to suppress quacks, and gave a commission to his minister Sinan to examine all practitioners, and license such only as he thought fit. As no less than 860 candidates presented themselves, Sinan's task was no sinecure, and we learn how popular medicine must have been at the time.

Another great physician of this Azudi or Jahya hospital was Ali ibn l'Abbas. He writes forcibly of the importance of students attending hospitals regularly with the professors, and noting the condition of the patients and all their symptoms, while at the same time they bear in mind the classical symptoms of disease as given in their text-books, and compare the two pictures. Indeed, medical studies were carried to a great height at this time. Not only did the leaders possess a good knowledge of Greek, Roman, and Indian writers, but by endless observations and case histories they built up a real advance in our knowledge.

In Moslem Spain new hospitals were added to the old ones at Toledo, Seville, and Algeciras, as well as at Cordova, where we find the Sultan Hachem regulating and reforming them, and appointing an examining board to license state physicians and the hospital staffs. Indeed, hospitals began to appear everywhere, from Merv and Mosul in Asia, Mecca and Medina in Arabia, Harran and Aleppo in Syria, to distant Fez in Algeria, Shiraz, Hamadan, and Ispahan in Persia—not to forget Saladin's foundation in Cairo.

Three of the greatest Moslem hospitals were at Tabriz, Damascus, and Cairo. The first of these was built by the great Vizier Rasidu in 1295. He sent agents to foreign countries to collect good or rare drugs, and built a huge model hospital, with a staff of surgeons, oculists, and bone surgeons, each served by five dressers; and thirty physicians, each having ten students as clerks. However, this had a short life, for his enemies in the troubles of the time succeeded in ruining him and destroying his beautiful hospital.

The Bimaristan at Damascus was built by the fierce warrior Nur ud Din after his victories over the Byzantines about 1160, and its comforts and splendours were open to rich and poor alike. This was more lasting, and for 300

years it is said that its fires were never extinguished. Here, too, was a great medical school, for which another block of new buildings was added in 1250.

It was when lying sick in this palatial hospital that a vow was made by the Sultan of Egypt, Kalaoun, that if he recovered he would build another in Cairo. The result was the noble Bimaristan which he opened in 1284. One still sees to-day its small but exquisite mosque and tomb chapel in perfect order. It had three courts with trees and splashing fountains, the chief court had a verandah into which opened the large wards, while the smaller courts were surrounded with little private ones. There were, indeed, wards for every disease known, laboratories, baths, lecture rooms, a dispensary, and a library with its five assistants. There were convalescent homes or allowances for those recovering, and an income of £25,000 per annum. At the present time part of this ancient house has been made into a very up-to-date eye hospital with male and female wards, operating theatre, X-ray and other installations, under the able care of Dr. Mohammed Khalil, late chief medical officer of the ophthalmic hospitals. Another fine Cairo hospital, El Moayad, now being restored by the State, did not arise till rather later, and must be omitted here.

It is difficult to classify these Moslem hospitals as to their founders. Most of them were due to military rulers, but individuals created many, and possibly some are due to bodies like Dervishes. Shrines, though existing everywhere and thronged by crowds of sick, have not, so far as I know, grown into hospitals.

To return to the European hospitals, in which remarkable changes began about 1100. We have seen that, besides endless foundations by individuals and some by princes, great numbers had been built and managed by bishops, canons, and monastic orders, and that, in the scarcity of lay physicians, canons and monks had largely monopolized medicine. The church found the drain too great, and that large numbers of her official staff were neglecting their proper functions, often for the sake of making money, to study and practise as secular lawyers, physicians, and pharmacists. It had to be stopped. Decrees by some ten councils and popes, 1131 to 1234, forbade the abuse, and the ecclesiastical practice of medicine was greatly reduced.[13] Fifty years later these stringent laws were relaxed under safeguards, but the check had been given. It is a curious thing that historians have often taken these councils as forbidding surgery to priests, which is never mentioned in the seven earlier ones, such as Tours. Only in 1215 does the prohibition creep in, to be relaxed with the others later on.

To fill the gap, besides the many lay doctors whom the fresh Universities and Schools soon provided, some 20 new societies of laymen to found and work hospitals were sanctioned. An enormous demand arose. It was the time of the epidemic of leprosy (1100 to 1350), the time of the Crusades, the date of a great religious revival, and of the revival of Guilds which were keen to take up any charitable work. Hospitals became the passion of the age. Individuals founded them everywhere or subscribed to an order of Hospitallers to do so.

Of these orders or hospital societies, three or four combined fighting with their work, and thus revived the military hospitals as well as civil ones.

1. *The Teutonic Knights*, besides their Palestine houses, founded sixty hospitals in towns in the Baltic area, such as Dantzic, Thorn, Elbing, and Königsberg.

2. *The Templars* founded others; for instance, those at Frankfort-on-Oder and Munchaberg.

3. *The Knights of St. John of Jerusalem* inherited an old hospital there, and about the year 1120 built a fine new one with 2000 beds under the care of five physicians and three surgeons, the former having to give proof of their skill in pharmacy and uroscopy. When the knights were driven out by Saladin they built other hospitals at Acre, Rhodes, Malta, and other places in Europe. Thus they took over and worked a large previously existing hospital at Salerno. Their house at Malta became perhaps the finest and best equipped hospital in Europe until it was plundered by Napoleon. Later writers tell of the patients in their snowy white cubicles, delicately fed, and served on silver plate.

4. *The Lazarists*, whose history is obscure, are said to have helped with leper hospitals, of which there were 200 in England and 1700 more on the Continent. I do not think that these were wholly quarantine stations. Treatment and even cures occurred, and it seems that these houses were sometimes used for other infectious diseases, so that the name of lazar house is then given to what we should call a fever hospital.

5. *The Order of St. Antony* met the outbreak of ergotism with houses in England, France, and Italy.

6. *The Society or Order of the Holy Spirit* was perhaps the most active of all.[14] Founded about 1170 by a Mr. Guy at Montpellier, it was so well managed that Innocent III gave it great privileges in 1198. It was a purely lay body of men and women for founding hospitals and nursing "*Our Lords the Sick*". By 1300 it had raised 900 hospitals. One branch worked in Hungary, Italy, and England. The Pope built for them the still existing Hospital of the Holy Spirit in Sassia at Rome on a site given by the English king where King Ina of Wessex had founded a rest-house 500 years earlier. The revenues of this hospital reached in after times the sum of £37,500 a year, and it is even now a fine building. The other province included France with 400 hospitals, certain other countries, and Germany, where Professor Virchow traced 155 hospitals of the Holy Ghost, their headquarters being at Montpellier. In England their records have been entirely lost, and though King John gave them an estate at Writtle, in Essex, and the bishops licensed them to collect alms, no hospital has been traced to them with certainty, although several bore that dedication, and one of these was amalgamated in 1436 with St. Thomas's in London. Besides their whole-time members, the Order had an enormous number of subscribers who raised funds and visited the sick, forming the Confraternity of the Holy Ghost. This had branches in Bristol and elsewhere, and in after days King Henry VII, the Lady Margaret, and John Colet were among its members.

I have not time now to speak of other societies of hospitallers—Albrac, Roncesvalles, St. Gervase, etc.—or to catalogue the many municipalities, guilds, and individuals who founded hospitals on the Continent at this time,

especially in such towns as Florence and Cologne. We may just notice that in England between 1100 and 1300 four hundred new hospitals and asylums were founded either by individuals, guilds and societies, or bishops and kings. This is in addition to the monastic infirmaries. Some of them were hospitals in the strictest sense, such as St. Bartholomew's, and we must not forget St. Thomas's in London, and those at Gloucester, Lincoln, Norwich, and Durham, and the older ones at York and Canterbury.

Finally, over Europe generally, since lay efforts had shown themselves so efficient, and since municipalities in some places had taken up public health as in Greek times, the decrees of the Councils bore fruit, so that a movement arose to hand over the management to lay magistrates. This was done in many large towns and in Rome itself, by the joint wish of all parties, about 1300, though somewhat later in France.

We have, then, briefly traced the growth of hospitals up to this epoch, just before the Black Death. One is struck with the great number of men of various races, religions, and civilizations who have laboured at the work. Then, when one thinks of the vast number of hospitals which have perished, regret must arise that the life of hospital systems is so fragile and so easily destroyed by war and by changes in fashion, in government, in religion, or medicine, and that such a huge volume of work has been thrown away by their early decay.

Is not this just what we find to-day, that invaluable as hospitals are, they are no sooner built than they are superseded, or need alteration? In fact they are typically "a short term investment".

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URETHRAL DIVERTICULA.

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URETHRAL diverticula may be congenital or acquired, and the causes of the acquired variety vary in the different portions of the canal.

In the prostatic urethra, pouches of this kind are most commonly acquired from the gradual increase in size, and communication with the urethra, of a sac or sacs containing calculi which formed in the gland substance.

In a second and less common variety of acquired diverticulum, the prostatic urethra may gradually become distended to form a smooth-walled sac as a result of the lodgement of a stone or stones in that portion of the canal; two of the three cases recorded by Morton,[1] and the following case published by Ferraton,[2] appear to have been of this kind.

The patient, age 24, a quartermaster in the Navy, had been treated for pain and difficulty in passing water, at the age of 4 years. Fourteen years later these symptoms recurred, and troubled him at intervals for the six years preceding the operation in 1904. An ovoid phosphatic stone, with a core of urates (25 × 18 mm., weight 5·697 grm.) was removed by the suprapubic route through the bladder, from a smooth-walled sac formed by the prostatic urethra.

Ferraton thought that the cavity was preformed and probably congenital in origin, because of the long history, and the disparity in size between the sac and the stone; but it seems more probable that the pouch was acquired about a stone which lodged in the prostatic urethra in infancy.

As rarer causes of prostatic pouches, it has been suggested that cysts or abscesses of the gland substance might burst into, and persist as acquired diverticula communicating with, the canal.

CONGENITAL PROSTATIC DIVERTICULA.

The congenital prostatic diverticulum is less common, and while several of the older writers described and figured prostatic diverticula which might well have been of this nature, the suggestion that a pouch in this region might be the result of the gradual distention of the sinus pocularis is of comparatively recent origin, and in our own literature appears first in an excellent paper on "Urethral Calculi, with Special Reference to Encysted Calculi of the Prostatic Urethra", published by Monsarrat[3] in 1912. While that author considered that the diverticula in his three cases were most probably acquired, from the gradual expansion and fusion of pockets which contained prostatic calculi, he discusses the possibility of congenital origin, and concludes that "it is impossible to negative the presence of a preformed diverticulum as the first stage of the whole process, in view of the records of such preformed pouches in this part of the canal".

Bourdillat's[4] thesis on *Calculs de l'Urèthre*, which appeared in 1869, contains the illustration of a prostatic pouch, here reproduced (*Fig.* 34), which might have arisen from distention of the sinus pocularis, for the position of the diverticulum, and the fact that it contains a single stone, are rather characteristic of the congenital variety.

FIG. 34.—Stone in prostatic pouch. (*From Bourdillat, loc. cit.*)

Bourdillat states that this illustration was borrowed from Crosse's *Treatise on the Urinary Calculus*,[5] but it does not figure in the copy which is now in the Royal Society of Medicine.*

It appears to be probable that the sac had thus arisen from gradual distention of the sinus pocularis in the following case, which has recently been under my care :—

Case 1.—**Two-ounce stone in sac which communicated with the posterior urethra.**

S. F., age 48, complained of increasing frequency and difficulty in passing water.

HISTORY.—He had served in the Royal Field Artillery from 1900 to 1919 ; in 1902 he had gonorrhœa, and was in hospital for six weeks and discharged as cured. Two or three years later he began to be troubled by pains in the perineum which were brought on by riding. His present symptoms have been noticed for the last two or three years, and have gradually got worse. He passes urine every two hours during the day, and about five times each night. " The water comes sharp at first, then stops sudden, and then starts again " ; and for some months he has been troubled by sharp pains above the left testis, which are brought on by coughing.

He has been married for ten years. No children, no abortions. There was no history of trauma ; no history of difficulty or incontinence in his youth, and there was a complete absence of any previous history of renal colic. Nothing abnormal had been noticed as regards his sexual functions.

He was sent up to out-patients as a case of stricture of the urethra, but on rectal examination a very large hard mass could be felt in the perineum, between the posterior urethra and the rectum, which was pressing on and obstructing the urethra.

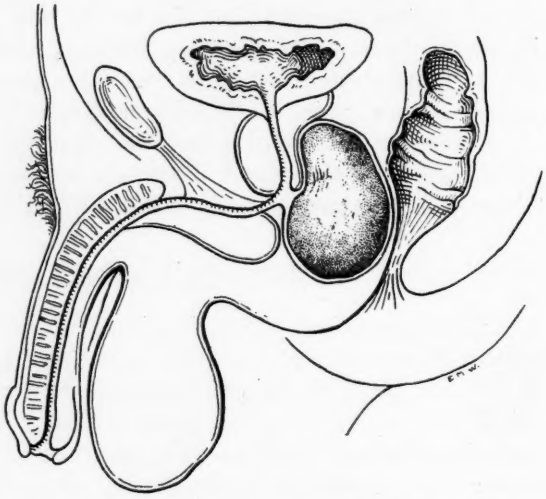


FIG. 35.—*Case* 1. Diagram of posterior urethral diverticulum and stone.

OPERATION.—He was admitted to hospital, and on Sept. 8, 1927, the hard mass was cut down on from the perineum, and proved to be a large phosphatic stone, 2 oz. 15 gr. in weight, occupying a large smooth-walled pouch which lay between the posterior urethra and the rectum, and communicated with the urethra near the apex of the prostate, by a median slit

* John Green Crosse was Surgeon to the Norfolk and Norwich Hospital, and published his work entitled *A Treatise on the Urinary Calculus* in 1835.

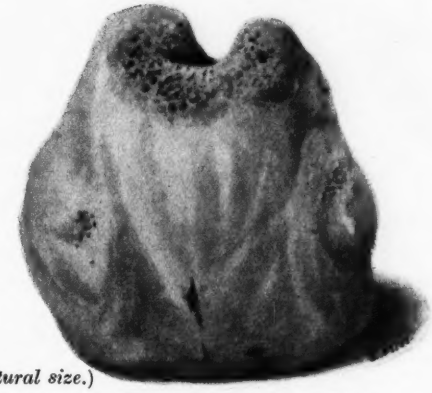
about half an inch in length (*Fig.* 35). The stone was deeply grooved on the surface which corresponded to the gap in the floor of the urethra, and formed a beautiful mould or cast of all the structures in contact with it (*Figs.* 36, 37).

The pouch was excised, and the edges of the aperture of communication with the urethra were stitched together over a gum-elastic catheter inserted into the bladder. The progress after operation was quite satisfactory. The perineal wound leaked urine for some days after the catheter was removed, but later healed, and at the present time a 10–13 bougie can be passed along the urethra into the bladder without any difficulty.

I am indebted to Dr. Guest, Pathologist to the Royal Infirmary, Sheffield, for the following histological report:

Sections cut through the wall of the diverticulum showed it to be composed of the following structures:

A lining membrane composed of definite stratified epithelium with well-marked prickle cells. This membrane is composed of two parts: a superficial, in which the



Case 1: Stone. (*Natural size.*)

FIG. 36.—Anterior aspect, with deep urethral groove.

FIG. 37.—Posterior aspect, moulded by pressure of rectum.

cells are of polygonal shape; and a deep part, where the shape of the cells becomes more elongated or cylindrical (*Fig.* 38). The most superficial cells are much flattened and their nuclei lost, and a tendency to keratinization can be seen in sections especially stained to show keratin. The nuclei of the deep cell layers took up hæmatoxylin much more readily than the superficial. Papillæ are well marked, and project upwards into the epithelium, and are composed of loose connective tissue.

Deep to the mucosa lies much fibrous tissue with scattered bundles of smooth muscle, the latter being more confined to the deep layers of the wall of the diverticulum, while blood-vessels are present in considerable numbers.

All layers show a round-celled infiltration, but this is more especially marked in the region of the subepithelial connective tissue. The vessels show some elastic tissue, and, throughout the fibrous and muscular tissue, fibrils of elastic tissue can be seen in suitably stained sections.

The stone which occupied the diverticulum has not been analysed, but on section it appears to be largely composed of phosphates, with some layers of urates, deposited around one central nucleus.

The grounds on which we base our hypothesis that the diverticulum was preformed, and had probably arisen from the sinus pocularis, which had persisted as a congenital defect that determined stone formation, and then enlarged *pari passu* with it, are as follows: the long duration of the history, and the complete absence of antecedent renal colic; the position, anatomical

relations, and histological characters of the diverticulum, and the fact that the stone was single, and had formed around one central nucleus. The

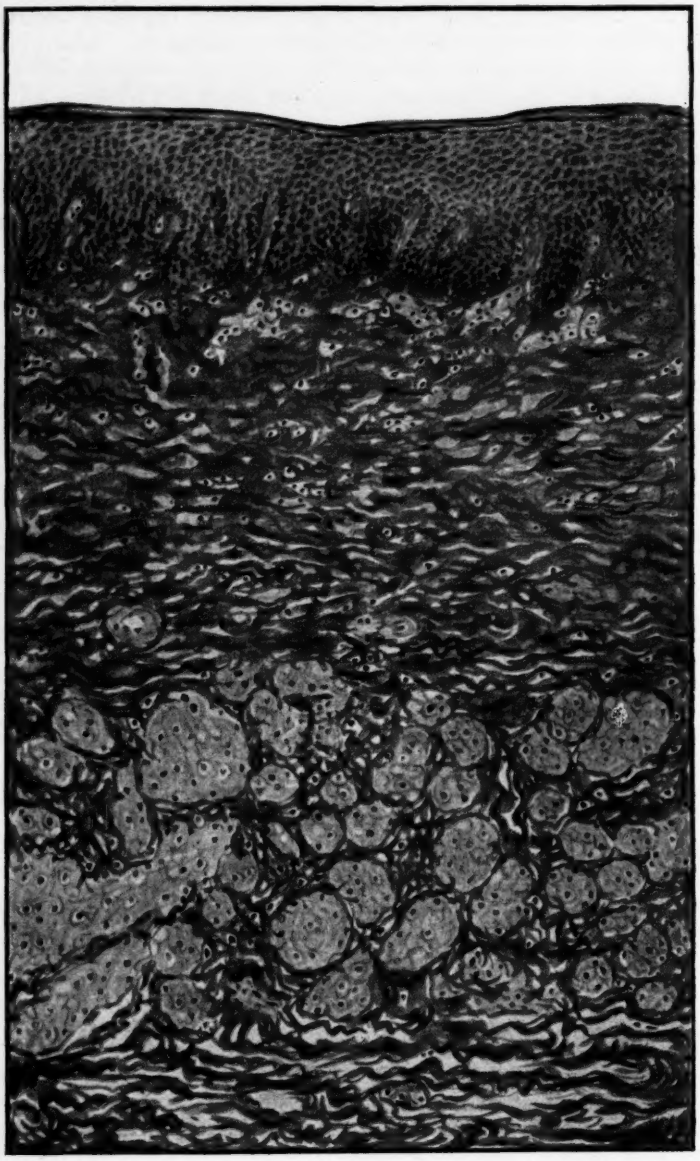


FIG. 38.—*Case* 1. Pen drawing of section of wall of diverticulum.

following somewhat similar cases, which have been recorded as examples of congenital prostatic diverticula, are cited in support of this opinion :—

Kapsammer[6] found in a man, age 30, who had experienced difficulty in passing urine from his youth upwards, a diverticulum of the prostatic part of the urethra, " exactly like a vaginal sac ", in which a phosphatic stone, which weighed 162 grm., had developed. On microscopic examination the structure of the wall of the sac was found to resemble exactly a section of the wall of the vagina.

Escat,[7] in an article entitled "Malformations Congénitales et Acquises de l'Urèthre," describes and figures (*Fig.* 39) a most interesting and instructive case of which he gives the following particulars: A young man, age 18, had noticed, from infancy, a swelling the size of a nut situated in the mid-line at the junction of the posterior aspect of the scrotum with the perineum. This swelling always increased in size when he passed urine, and he used to press on it to empty it by the urethra. A few days before he presented himself he found that he could no longer empty the swelling, which became bigger and painful. The diagnosis of an inflamed congenital urethral pouch was made, and, after the pouch had been incised and emptied of pus and urine, an attempt was made to dissect out the wall of the sac, which narrowed down to a pear-shaped neck, from which a hollow stalk ran along the right side of the bulb, through the triangular ligaments, to end in the pars prostatica. A section of the stalk of the sac showed that it was lined by mucous membrane, enclosed in a sheath of unstriped muscle fibres. The mucosa comprised an epithelium and a dermis. The epithelium was stratified and resembled skin, with polygonal and cylindrical cells in the deeper layers; while, however, in the stalk the superficial layers showed no corneous change, and few and ill-formed papillæ, in the walls of the dilated sac the superficial cells showed keratinization, and there were well-marked papillæ in the dermis. There were no glands. The dermis was composed of adult connective tissue, with, in places, aggregations of lymphoid cells such as one finds in the walls of certain ranulæ and congenital branchial fistulæ. Outside the dermis there was a layer of unstriped muscle, and the stalk was enclosed by a fibrous sheath, which was firmly adherent to the layers of the triangular ligament.

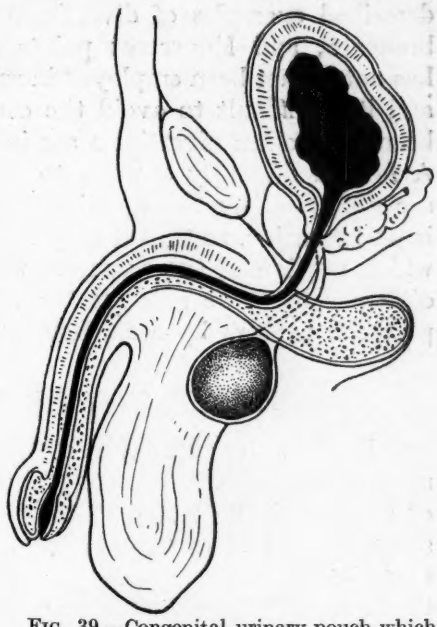


FIG. 39.—Congenital urinary pouch which communicated with the posterior urethra. (*From Escat, loc. cit.*)

Escat remarks that the above is an unusual example of congenital malformation, as the urinary pouch was developed slowly at the inferior extremity of a canal nearly 10 cm. in length. The hollow stalk was about the size of the round ligament, and measured 2 mm. in diameter, while the urinary pouch was about the size of a nut, and situated in the base of the scrotum. The author was unable to find a record of any similar congenital defect, but suggests that his case might be analogous to certain very rare congenital perineal fistulæ, two examples of which are referred to by Legueu in Le Dentu and Delbet's *Treatise on Surgery*; in both there was a tiny median fistula, which allowed a few drops of urine to escape at each act of micturition, and the fistulous tract, which was lined by epithelium, and enclosed in unstriped muscle, communicated only with the posterior urethra. Escat concluded that his case was a variation of the same malformation, and that the anatomical relations and histological structure suggested that it was a persistent portion of the Müllerian duct, which forms the vagina in the female.

Another probable example of a congenital urethral pouch, somewhat similar to Escat's case, was described by Thistle[8]:—

A man, age 32, who had been treated for some years for a stricture of the urethra, was admitted to the Torbay Hospital, complaining of pain and swelling in the perineum, and of inability to pass urine. The perineal phlegmon was incised, and the central fluctuating swelling proved to be a very large inflamed urinary pouch, which extended into the perineum, and backwards behind the base of the bladder, and contained three uric acid stones which weighed 91, 124, and 425 gr.; the largest stone filled the back of the pouch behind the neck of the bladder, and had to be extracted with lithotomy forceps. The walls of the perineal pouch were rough and sloughy, and Thistle thought that it "was most probably formed by the dilated urethra behind the stricture and the walls of an abscess". The stricture was in the bulbous portion of the urethra, and admitted a catheter some weeks later, "when the parts had been kept at rest and all element of spasm has disappeared". The perineal wounds healed, but the patient was left with a perineal urinary fistula.

In addition to the cases quoted above, Finsterer[9] and Schapiro[10] have described examples of diverticula which communicated with the pars membranacea, and Monsarrat points out that the term 'membranous urethra' has not in fact been employed in so strict a sense by surgeons as by anatomists, and it is difficult to avoid the conclusion that some of these so-called membranous urethra diverticula are in reality diverticula of the prostatic urethra. A further difficulty appears to lie in the fact that the anatomical relations are disturbed by the growing pouch; the prostatic substance atrophies or is pushed aside, so that it may be a matter of real difficulty to determine into which portion of the posterior urethra the neck of the sac opens, and this difficulty must be still greater where the sac only communicates with the posterior urethra by a tubular stalk as in Escat's case.

DIVERTICULA IN THE ANTERIOR URETHRA.

In the anterior urethra secondary or acquired diverticula are again much more common than the congenital variety; but where the complete absence of any previous history of colic appears to exclude the possibility that the stone descended the urinary tract to become lodged in the urethra, and when there is no stricture or other local lesion likely to give rise to urethral or paraurethral stone formation, the possibility that a pouch of this kind may have originated from a congenital defect in the formation of the urethra must be considered, and appears to be probable in the following case:—

Case 2.—**Stone in pouch which communicated with the floor of the urethra at the peno-scrotal junction.**

H. A., age 48, complained of gradually increasing difficulty in passing water. There is no frequency, but the stream has been gradually getting smaller in size, and for some months he has had to strain to pass it.

History.—The patient first noticed a hard lump under the base of the penis in 1917, but did not trouble about it till his present symptoms developed. He is married, and has three children, the youngest four years of age. There is no previous history of trauma, or of renal colic. He has never had retention, incontinence, or urinary symptoms of any kind before the gradual onset of his present difficulty. He denies ever having had gonorrhœa.

On Examination.—There was a hard rounded swelling about the size of a walnut in the mid-line at the penoscrotal junction, which on passing a bougie was found to be caused by a stone lying in a pouch, which opened into the floor of the urethra.

Operation.—The patient refused to come into hospital, so on Sept. 9, 1927, the stone was cut down on, under local anæsthesia, and removed from the thin-walled

sac, which was tightly stretched over it. The small median aperture of communication of the pouch with the urethra was stitched up, and the thin-walled sac was obliterated by suture to form a pad, under the repaired floor of the urethra.

The position of the sac is shown in the accompanying diagram (*Fig.* 40). The stone weighs 158 gr.; the middle layers are composed of oxalates, while the outer and inner portions are phosphatic (*Fig.* 41).

In 40 cases of acquired diverticula of the urethra collected by Bourdillat, there was a history of some previous operation on the urethra (external urethrotomy, median or lateral lithotomy) in no less than 16, while in the remainder, false passages, blows or falls on the perineum, peri-urethral abscesses, and lastly all solutions of continuity of the canal such as 'crévasses spontanées' and 'poches urineuses' are mentioned as possible causes of the condition. Bourdillat, unfortunately, does not elaborate his suggestion that a urinary pocket might be the primary and determining cause for stone formation outside the urethra, and it was left to later writers to suggest that urethral diverticula might arise as a congenital malformation from defective fusion of the inner genital folds which form the floor of the anterior urethra.

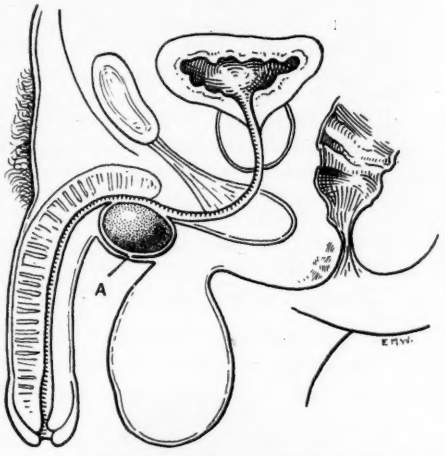


FIG. 40.—*Case* 2. Diagram of stone in urethral diverticulum (A).

FIG. 41.—*Case* 2. Stone. (*Natural size.*)

Escat describes a congenital pouch of this kind, which opened into the floor of the penile urethra in a child of 5 years, and points out in discussing this case that all varieties of the defect in the urethral floor may be met with, from the gross malformation of hypospadias to minor defects such as blind internal or complete fistulæ or dermoids of the median raphe.

Erichsen[11] writes on the subject of urethral calculi: "In other instances they appear to be formed in a pouch that lies to the outside of the urethra, and that is only connected with it by a small aperture . . . one of the most remarkable instances of this kind is represented in the annexed cut (*Fig.* 42), taken from a drawing in Sir R. Carswell's collection at University College. The stone here was of very large size—equal in bulk to two horse chestnuts".

Hurry Fenwick[12] describes a similar case:—

A large stone, which measured 3⅛ in. long, and 1 in. diameter, was removed by perineal section from a pouch in the floor of the penile and spongy urethra

in a man of 67 who had had difficulty in passing urine for eight or nine years. The anterior end of the stone was 2½ in. from the external meatus, and only the nose of the stone projected into the canal.

In recent years Boeminghaus[13] recorded the following case :—

A man, age 36, who had suffered from retention at the age of 9, and a year or two later noticed a hard pea-like swelling on the under surface of the penis, which had recently become painful and swollen, and discharged urine and pus from

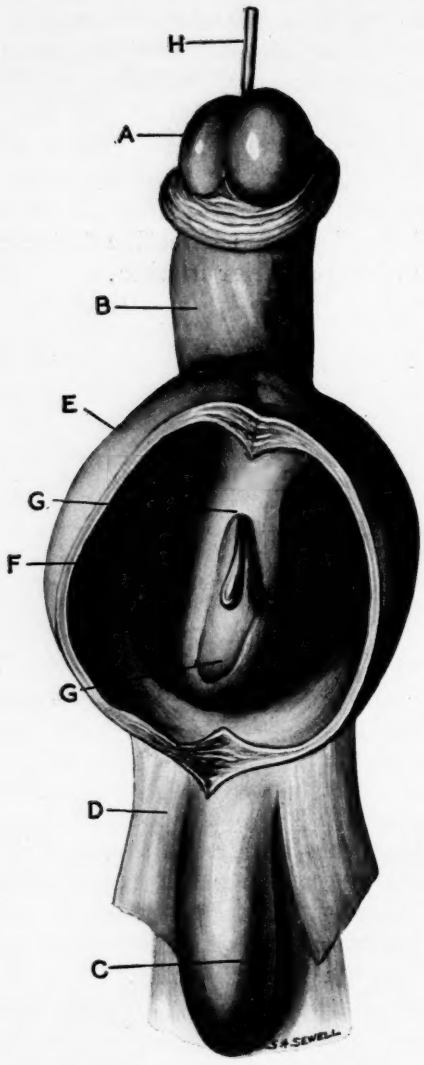


FIG. 42.—Dilatation of urethra in a case in Sir Robert Carswell's Collection. A, Glans penis; B, Body of penis; C, Bulb of the urethra; D, Crus penis; E, Wall of the dilatation, formed superiorly by the corpus spongiosum, laterally and inferiorly by the skin and cellular tissue; F, Cavity of the dilatation; G, G, The extent of the urethra communicating with the cavity. The probe (H) is introduced to render the direction of the urethra more apparent.

This cavity contained the calculus, and although the sound had been frequently introduced its presence had never been detected, probably because of the situation of the dilatation, which occupied the inferior surface of the penis, and because the stone did not project into the urethra. The patient was treated for sarcocele in the Hôtel Dieu, Paris.

The stone is shown separately.

fistulous openings. The swelling was incised, and a phosphatic stone, which measured 6 × 5 × 4 cm., and weighed 136 grm., was removed from a large congenital sac lined by stratified epithelium, with well-marked papillæ in the dermis, outside which was a layer of unstriped muscle enclosed by a fibrous tunic. The upper surface of the stone was deeply grooved by the urethra. The diverticulum opened into the floor of the urethra at the penoscrotal junction, by a median slit-like aperture 2 to 3 cm. in length. The sac was excised, and the edges of the opening into the urethra were sutured.

The description of a case which closely resembled the above was published by Neugebauer[14] in 1924 :—

> The patient, a miner by occupation, was 20 years of age. A small swelling, first noticed at the base of the scrotum when he was a year old, gradually increased in size, and later small fistulæ formed in the right side of the scrotum, and discharged urine during micturition, at which time two swellings as big as plums appeared at each side of the penoscrotal junction. After the flow of urine ceased, he still felt a desire to pass water, which was relieved when he squeezed these swellings, and thus emptied them by the urethra and the scrotal fistulæ. Fourteen small phosphatic stones were removed from a diverticulum, about the size of a hen's egg, which opened into the urethra by a median slit-like aperture 1·5 cm. in length ; while the lower end of the sac communicated through a narrow opening with a separate compartment, situated in the base of the scrotum, which contained another phosphate stone as big as a plum. Both the sacs were lined by stratified epithelium.

COMPOSITION OF URETHRAL AND PARA-URETHRAL STONES.

This has given rise to considerable discussion, for, while it is agreed that stones formed in the prostatic substance are composed of calcium phosphate, calcium carbonate, and organic material, and that any of the ordinary kinds of calculi may descend the urinary tract, to lodge secondarily in the urethra, there is by no means a similar unanimity of opinion as regards the composition of stones formed primarily in urethral diverticula, as the view expressed by several writers, backed by the authority of Englisch,[15] that stones formed in diverticula must necessarily be composed wholly of phosphates, is opposed by other surgeons.

It is interesting to note that in 1835 Crosse pointed out that calculi formed in the prostatic substance might subsequently acquire deposits of urinary salts, if the pouches which contained them became so enlarged as to communicate with the urethra ; while Monsarrat concludes that " given a preformed diverticulum, and the urinary constitution which tends to the production of calculi, there does not appear to be any difficulty in assuming that calculi other than phosphate may be formed *in situ* ".

Our two cases appear to support the latter view, as the large stone (*Case* 1) contains some layers of urates, while the middle layers of the stone from the pouch below the anterior urethra (*Case* 2) are composed of oxalates.

DIAGNOSIS.

The diagnosis of urethral diverticula embodies two main problems : (1) The determination of the presence of a diverticulum ; and (2) Its nature ; and while it is frequently possible to decide the first from the clinical symptoms, which vary with the position, size, and contents of the sac, the second question can more often be settled only after operation and histological examination of the wall.

The diagnosis of a pouch opening into the anterior urethra is often fairly obvious, as the stone or stones which are usually contained in the sac can generally be felt in the perineum, or where the sac is not fully occupied by calculi it may become distended during the act of micturition, so that the

patient may have to press on the swelling to empty it through the urethra, or from the fistulæ which result from the septic infections which so frequently supervene in and around these diverticula communicating with the anterior urethra. The congenital diverticulum in this region may form a swelling which dates from infancy, while the causes of the various acquired pouches are usually determined by the presence of a stricture, or by a history of colic, operation, local injury, or disease.

The diagnosis is more difficult where the diverticulum is connected with the posterior urethra, though the surgeon to-day has the advantage that it may be possible to fill up a urethral or prostatic pouch with lipiodol or sodium bromide so that it will show in a radiogram; and in all cases the calculi, which are almost invariably present, may be thus demonstrated, even when they are embedded in the prostatic substance. In the patient of whom the skiagram is given (*Fig.* 43), the prostatic concretions had been thus observed to grow, till the stones in one lateral pouch came to communicate with the urethra, and it became necessary to remove them from the atrophied prostatic shell by median perineal section, because the patient got retention, and the spasm and intolerance to instrumentation, which, as Monsarrat pointed out, is such a troublesome feature in this type of case.

FIG. 43.—Skiagram of prostatic calculi.

According to Erichsen, calculi in the prostate give rise to "a sense of weight, pain, and irritation in the perineum, sometimes to retention of urine, and in fact to the ordinary symptoms of enlarged and irritated prostate"; while Crosse wrote as follows on the same subject: "When small and thus embedded in the ducts, prostatic concretions cause none of the characteristic symptoms of urinary calculi and cannot be detected; it is only when they increase and rise so as to project at the orifices of the prostatic ducts, or escape as they may do into the urethra, that they can be detected by the sound; but when of large size, or when numerous and contained in one cyst, they can be detected by the finger pressing on the gland through the rectum. . . . The pouch enlarges in the direction where there is least resistance towards the lateral or posterior surface of the prostate gland; thus in extreme cases the concretions are felt per anum lying close to the rectum, and in a cavity no longer communicating with the urethra".

The curious crepitation which may be felt by the examining finger is very characteristic, and has been compared to 'beads in a bag'.

When the diverticulum has been gradually acquired around a stone lodged in the prostatic urethra, there may be an old history of colic or bladder trouble, and the stone can usually be felt by a sound, while in other forms of diverticula a stone can only be detected in this way when it grows large

enough to project into the canal, or when a small calculus passes from the pouch into the urethra, where it may become impacted. In more than one of the recorded cases of prostatic pouches containing stones, this complication occurred, and when the stone was cut down on from the perineum it was found to have disappeared in a puzzling manner, as it had slipped back into the prostatic diverticulum.

TREATMENT.

The treatment of urethral diverticula necessarily varies; but where it is possible an attempt should be made to dissect out the wall of the sac, after it has been opened and emptied of its contents, while the aperture of communication with the urethra is closed by suturing lateral flaps, which have been dissected up at the neck of the sac, over a gum-elastic catheter passed along the urethra and left tied into the bladder.

This ideal method of treatment is generally possible—in the absence of any acute infection—in all varieties of diverticula of the anterior urethra, and also in the congenital prostatic pouch; whereas the most that can be done in the acquired forms of prostatic diverticula is to cut down and evacuate their contents, by a median perineal section. The stones are liable to re-form after this method of treatment, and for that reason Morton had to repeat the operation on one of his patients on six separate occasions.

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DIVERTICULOSIS OF THE APPENDIX AND PSEUDOMYXOMA PERITONEI.

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DIVERTICULOSIS OF THE APPENDIX.

A. J. GARDHAM.

The condition of diverticulosis of the appendix has received very little attention in English medical publications, although there are numerous references to the subject in the literature of other countries. It is for this reason that it seems appropriate to place the following case on record.

FIG. 44.—The appendix after separation of the omentum. A. Intact diverticulum; B, Ruptured diverticulum.

The patient, a man age 34, was admitted to University College Hospital under Mr. E. K. Martin on Jan. 25, 1927. The following particulars are taken from the case notes.

HISTORY. — Eleven weeks before admission to the hospital the patient had a sudden attack of pain in the lower abdomen, accompanied by nausea. The pain lasted some hours only, and then passed off, leaving nothing more than a feeling of soreness. Similar attacks recurred at intervals of two to four weeks. The patient was admitted to hospital in the fourth attack, eleven weeks after the first symptom. There was no history of abdominal pain previous to the present series of attacks.

PRESENT STATE.—The patient is a healthy-looking man. Tongue clean and moist. Temperature 98°–98·8°; pulse 80. Abdomen: There is a slight visible swelling in the right iliac fossa. On palpation a hard, slightly tender mass is felt, attached to the anterior abdominal wall. The mass does not extend to the limit of the peritoneal cavity laterally. Internally it appears to pass over the brim of the pelvis. Per rectum, a mass can be felt high up on the right side of the pelvis.

OPERATION.—Muscle-splitting incision over the right iliac fossa. The mass exposed consisted mainly of the great omentum. On separating the

great omentum the appendix was exposed lying to the outer side of the cæcum. The appendix, which showed the condition described below, was removed, together with the omentum involved in the mass. No pus was encountered at any part of the operation. A drainage tube was left in the wound on account of the rather marked oozing of blood which followed the separation of the omental adhesions. The patient made an uneventful recovery and left the hospital on the fourteenth day.

After separation of the omentum the appendix showed the following condition (*Fig.* 44). In its proximal three-fourths the organ was of normal size. At the junction of the third and distal fourths was a spherical reddish projection the size of a pea, attached to the antimesenteric border. The distal fourth of the appendix was distended, and on its lateral side about one-third of an inch from the tip was a projection of mucous membrane, there being in this situation an opening from the surface into the lumen of the appendix. It is probable that this opening into the second diverticulum was the result of trauma in separating the appendix. The lumen of the appendix proximal to the diverticula was still patent, but so much diminished in size that an ordinary probe could not be passed, the patency being demonstrated by passing a fine bristle through it. The omentum removed with the appendix was very fibrous, but showed no mucoid material.

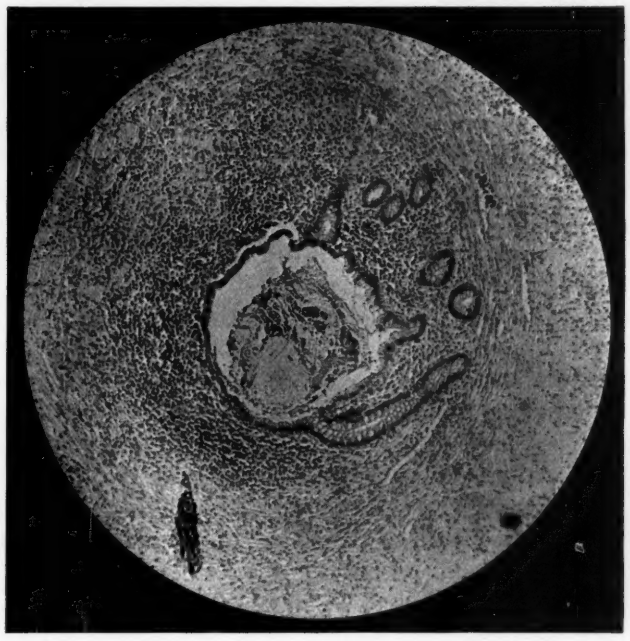

Fig. 45.—Section of the appendix proximal to the diverticulum, showing contracted lumen. (×56, $\frac{2}{3}$ obj.)

Microscopic Sections.—

1. The unruptured diverticulum shows a clean-cut gap in the muscularis, through which the diverticulum protrudes. The diverticulum consists of a single layer of mucous membrane (as opposed to a number of cases quoted by Stout[1] in which the diverticulum consisted of a hernia of the complete mucous lining of the appendix). The covering of the diverticulum consists mainly of granulation tissue, a little fibrous tissue, and a few muscle fibres. The peritoneal coat has disappeared over the greater part of the diverticulum, and in these 'raw' areas numerous newly-formed vessels can be seen, as if the tissues of the diverticulum were engaged in picking up a new blood-supply from the surrounding omentum. The mucous membrane of the diverticulum is seen, on

staining with mucicarmine, to be actively engaged in the production of mucin.

2. The surrounding omentum shows numerous strands of fibrous tissue. Mucicarmine reveals no mucus.

3. A transverse section of the appendix proximal to the diverticulum shows a definite but extremely contracted lumen. The diminution in the lumen is due to an increase in the fibrous tissue in the submucous coat (*Fig.* 45).

COMMENTS.

The points which I wish to emphasize in this record and which I believe to be characteristic are, firstly, the history of repeated attacks of very slight intensity, and secondly, the presence of a fixed mass in the appendix region in the absence of any of the ordinary signs of inflammation (compare with Neumann's[2] and Trotter's[3] cases).

The main interest of diverticulosis of the appendix lies in the relation of the condition to pseudomyxoma of the peritoneum. Diverticulosis of the appendix appears to be a condition of only moderate rarity; in addition to the case here described there are three appendices showing diverticula in the Museum of University College Hospital. Moschcowitz[4] found the condition in only 4 of 1500 appendices investigated, and McCarthy and McGrath[4] in 17 of 5000; but investigations by Stout appear to show that diverticulosis is of considerably greater frequency than this. It can, I think, be said with certainty that diverticulosis of the appendix is a much less rare condition than pseudomyxoma peritonei. On the other hand, those cases of pseudomyxoma peritonei which have originated from the appendix have been shown in nearly all cases in which the appendix has been fully investigated to be associated with diverticula; according to Neumann, 4 of the 8 cases of pseudomyxoma peritonei investigated by him showed diverticula of the appendix. It appears, then, that diverticula of the appendix frequently, but not necessarily, lead to a pseudomyxoma of the peritoneum. It remains to decide the additional factor which must be present for a diverticulum to lead to pseudomyxoma peritonei.

Two recorded cases of early pseudomyxoma peritonei, those of Neumann and Trotter, bear a very close resemblance to the case here recorded. All three gave a history of recurring mild attacks, and all three presented physical signs of the same type, namely, a mass in the right iliac fossa resembling the ordinary appendix abscess, but unassociated with fever, rapid pulse, or constitional disturbance. In the case here recorded this combination of marked physical signs with mild symptoms was noted, but was thought to be accounted for by marked encapsulation of an abscess. In Neumann's case, as in my own, the mass consisted of omentum, and the appendix presented an unruptured diverticulum. In Neumann's case the lumen of the appendix proximal to the diverticulum is not known to have been obliterated; in mine it was definitely patent, although this could only be conclusively demonstrated by a microscopic section.

Recorded cases of diverticula of the appendix nearly always give a history of repeated attacks, usually of slight or moderate intensity, but in many an attack of greater severity led to the removal of the appendix (Chase,[4] Stout[1]).

It appears that the life-history of a diverticulum of the appendix is as

follows: One or frequently more diverticula are formed as a result of destruction of small areas of the muscle coat of the appendix by interstitial abscesses in the course of an attack of appendicitis. This destruction of muscle is probably a frequent event, but in the majority of cases the mucous membrane is involved in the destructive process, and the result is the ordinary 'perforated appendix'. If the mucous membrane is not destroyed a diverticulum results. In the majority of cases repeated attacks of inflammation lead either to perforation of the diverticulum and abscess formation, or to operative removal of the appendix.

In a minority of cases the inflammation dies down sufficiently to allow perforation of the diverticulum to occur without leading to abscess formation. These are the cases which ultimately develop pseudomyxoma peritonei. The actual changes which immediately precede the formation of the pseudomyxoma are not known, but the following theory appears reasonable to me.

The presence in many of the recorded early cases of an omental mass in the region of the pseudomyxomatous appendix, and the fact that pseudomyxomatous nodules in the omentum have come to be regarded as a characteristic sign in the early stages of the disease, suggest together that the omentum is closely connected with the production of pseudomyxoma peritonei. In the case here recorded the mucous membrane, in part at least of the diverticulum, seems to obtain its blood-supply from the omentum. Supposing rupture of the diverticulum to take place, it is possible that some part of the mucous membrane having already established a blood-supply from the omentum would remain to lead a parasitic existence outside the diverticulum. The logical sequence of this would be the continued production of mucus after removal of the appendix, a condition which has been recorded but not explained by those dealing with the fully developed condition of pseudomyxoma peritonei. The frequent absence of an epithelial lining in the diverticula in cases of pseudomyxoma peritonei also suggests that the rôle of the diverticulum is not an active one in the later stages of the disease.

I am indebted to Mr. E. K. Martin for permission to publish the case, which came under my care when acting as deputy for him.

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SOME SHORT NOTES ON PSEUDOMYXOMA PERITONEI.

C. C. CHOYCE.

THIS condition in association with perforated ovarian cysts was first described by Werth in 1884, and since then has been noted by many others, but it was not until 1901 that Fraenkel described the first case in connection with a perforated cystic appendix. This was found, unexpectedly, at a post-mortem examination in a man of 79 who had died of cerebral softening.

Emil Ries reviewed the literature in *Surgery, Gynecology, and Obstetrics*, in November, 1924, p. 569. His summary shows that although all the early cases were in women, and associated with ovarian cysts, quite a number of the later ones were found in both men and women, and in connection with cyst formation in the appendix, or sometimes with diverticulosis of such cystic appendices.

In 1910 Trotter (*see above*) found in the literature 9 cases of appendicular origin and added another of his own. He found that three varieties of associated appendicular abnormality were to be seen: (1) Simple obstruction resulting from fibrosis (or, in one case, from carcinoma of the appendix); (2) Diverticula of mucous membrane through the appendicular wall, and subsequent rupture; (3) Multiple cystic degeneration of the wall of the appendix. In this connection it is interesting that an appendix removed in the University College Hospital recently showed diverticulosis, the diverticulum containing a mucoid material. This case is briefly recorded and illustrated above by Mr. A. J. Gardham. It is possible that if this diverticulum had ruptured, the condition of pseudomyxoma peritonei would have been set up.

It is a curious fact that some of the cases have been found to be associated in the same patient with both ovarian cysts and cystic appendices; moreover in at least one case there was a mucoid collection in an umbilical cyst due to non-obliteration of the omphalomesenteric duct.

In some of the cases in which a cystic appendix was also found, no communication with the interior of the appendix was demonstrated, but in Bailey's case recorded in *Surgery, Gynecology, and Obstetrics*, 1916, p. 219, there were perforations of both the ovarian cyst and the appendix. In many of the cases in which the appendix only was at fault, there was an actual perforation; in some there was evidence that perforation and subsequent closure had occurred.

The jelly-like material is variously stated as mucin or pseudomucin, and gives the mucin reaction with thionin; it is of the same character whether it originates from the appendix, the ovary, or both.

It would be supposed that removal of the source or sources, and removal of as much of the pseudomyxomatous material as is possible, would cause cessation of its development, but this is by no means always so, for several patients have had to be operated upon more than once. The jelly-like material, therefore, must either be capable of reproducing itself, or of being reproduced by cells that have been dislocated from ovary or appendix and implanted elsewhere in the peritoneal cavity. Careful search has been made for such cells by several writers, who have, however, been disappointed. No one has yet demonstrated any very definite clump of such cells, though in some cases chains of cubical cells have been found (as in Trotter's case). For the present it seems fair to assume that epithelial cells of this type may be the cause of recurrence. If that be so, there seems to be some evidence that eventually they die out, for several patients are alive and well a considerable time after repeated operations.

As regards the condition itself, it is difficult to improve upon Werth's description in 1884 as translated by Ries. He speaks of the "peritoneal cavity being filled with gelatinous material in its whole extent. This material is partly free and partly in the form of thick semitransparent layers lying

on and firmly attached to the abdominal wall and intra-abdominal organs. The masses contain delicate connective-tissue membranes and fine vessels. At times the gelatinous substance is enclosed in a delicate connective-tissue membrane, and forms, especially on the intestine, polypoid pedunculated structures". These statements exactly describe the conditions found in Dr. Randall's case noted below.

In many cases the peritoneal endothelium is intact under the adherent masses; in others endothelial cells seem to have grown over the masses as well.

As regards treatment and prognosis, it would appear that the ovary and appendix should *both* be suspect, and that if not healthy both should be removed; the coexistence of a cystic appendix with an ovarian cyst has been sufficiently common to cause certain writers, without a great deal of justification, to speculate that either the ovary was secondary to the appendix or vice versa.

If all the gelatinous material can be removed without too much damage to the peritoneum upon which it is superimposed, this should be done; and in view of certain reported cases of pseudomyxoma arising from an umbilical cyst, it would be wise to examine the umbilical region also. No drainage of the peritoneal cavity should be done. Many of the early cases in which this was done died. Recurrence may take place, and second or even third operations may be necessary, but the general tendency nowadays appears to be towards survival of the patient if the case is seen sufficiently early for proper surgical treatment. In the case quoted above this stage had long been passed.

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PSEUDOMYXOMATOUS CYST OF THE PERITONEUM FOLLOWING APPENDICITIS.

MARTIN RANDALL.

J. F. T., male, age 71, was admitted to Nelson Hospital, Merton, on May 25, 1927. The complaint was of general weakness and great abdominal enlargement, with intolerable discomfort and distress.

HISTORY.—About three years ago patient had an illness, with abdominal pain of fairly acute onset for which no cause could be found. He was not gravely ill, but since then his abdomen has gradually enlarged. During these three years he has been an invalid, spending his time in house or garden, and much of it in bed. His appetite has been fair, and bowels regular.

On Admission.—Patient is pale and listless. He walks slowly and with difficulty, but can stand upright. There is no obvious difficulty in breathing. No sign of disease is found in mouth, lungs, or heart, and the urine is normal. There is no œdema of legs. Pulse 76; respiration 22; temperature 97·6°.

The abdomen is enormously and uniformly enlarged. The skin is not shiny, nor is the tension very great. The enlargement is such as to cause a direct forward projection of about three inches from the costal margin—the lower ribs being widely opened out. The whole abdomen is dull, and no resonance can be detected, even in the flanks. A fluid thrill is everywhere perceptible, but it is a somewhat sluggish thrill. Nothing could be felt on rectal examination beyond a general fullness of the pelvic cavity. An exploratory puncture made in the middle line below the umbilicus with a large abdominal trocar, only succeeded in obtaining a little gelatinous fluid, too thick for any considerable quantity to run through the trocar.

No diagnosis had hitherto been made, and this brought us no nearer one. At this stage Mr. Wilfred Trotter was kind enough to see the patient. He made a diagnosis of a myxomatous cyst originating from the appendix, and advised an exploratory operation.

Operation.—This was undertaken by me, on May 31, 1927, with the help of Mr. C. M. Brophy. A right paramedian incision with centre opposite the umbilicus was used. The cyst wall and peritoneum could not be differentiated, and a huge cavity was opened, full of thick stringy mucoid material, which did not run out freely, but had to be squeezed out. There were about 40 pints of the material. No bowels or viscera of any kind could be felt in the cavity, which proved to be that of a huge cyst. The patient was very far from well, so it was judged impossible to remove the cyst and it was closed, drainage appearing to offer no reasonable ground for hope. The patient gradually sank, and died on June 5.

Post-mortem.—An abdominal examination revealed the presence of a huge cyst, with a thick tough wall, the inside of which was almost black in colour. It extended to every recess of the abdomen and right down between the bladder and the rectum. Its connections with the surrounding structures were very intimate, and much force had to be used to remove it: it would have been quite impossible to remove it at the operation without a fatal issue on the table. The point of origin appeared to be in the neighbourhood of the cæcum; the specimen was sent to Mr. C. C. Choyce for investigation.

Description of the Specimen.—The specimen is shown overleaf in *Fig.* 46. Allowing for the reversal of right and left due to the fact that it is viewed from behind, it will be seen that the cyst fills the whole space from the ascending to descending colon laterally, and from the transverse colon to near the brim of the pelvis below. The posterior wall of the cyst has been largely removed. The cæcum is free from the cyst wall, and from it runs the appendix, also free up to a point an inch from its tip; this distal portion of the appendix passes through the cyst wall, and the tip of the appendix can be seen free, but lying amongst a mass of inflammatory tissue inside the large cyst. It is perforated at its tip. The inner aspect of the cyst wall is brownish in colour and much corrugated; it is everywhere coated with a

yellow grit. The assumption is that the contents of the cyst represented the secretions of the appendix over the years since his first complaint of symptoms. The grit on the cyst wall is composed of cholesterin, and the

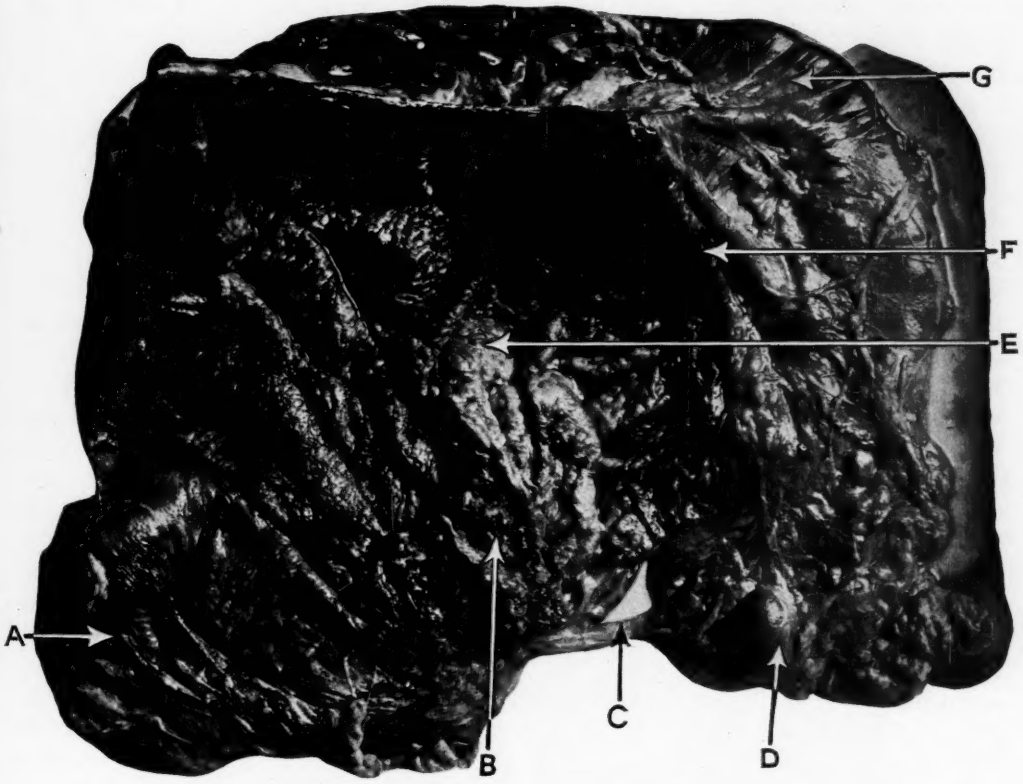

FIG. 46. — Pseudomyxoma peritonei arising from appendix (seen from behind). A, Sigmoid colon; B, Tip of appendix in mass of inflammatory tissue inside cyst; C, Appendix; D, Cæcum; E, Much corrugated lining, coated with yellow grit; F, Cut wall of cyst; G, Region of hepatic flexure.

gelatinous contents of the cyst gave a positive mucin reaction with thionin. Microscopically it was almost structureless, except for a little very delicate connective tissue, especially near the edges of a mass.

ON THE VEINS OF THE DIPLOË.

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THE existence of venous channels in the interior of the various bones of the human skeleton is to-day so widely, so universally recognized, that it seems strange that any era of anatomical knowledge can have existed when men went in ignorance of them. That the bones were well supplied with blood was certainly known, as the operation of amputation must have early taught the surgeon-anatomist. But that there were definite venous channels, that ordered systems existed in the interior of the bone, and particularly that these were present in the diploë and vertebræ, were certainly unknown until the earliest years of the nineteenth century.

In the year 1803 Dupuytren published in Paris a small monograph the first chapter of which is entitled "Canaux Veineux des Os." In this section he speaks of the little-known veins of the diploë as difficult or impossible to inject, but possible of display by section of the bone (as in the vertebræ) or by chiselling away the compact outer layer (as in the skull). Dupuytren gave a brief summary of the diploic veins, stating that there are three or four channels in the bone on each side draining from the summit downwards, and emptying into three sets of veins, namely, the external veins, the veins accompanying the meningeal arteries, and the meningeal sinuses of the cranial base. There is very little detail, and it is evident that Dupuytren is satisfied to be placing on record the bare outline of a newly discovered fact.

Here the matter rested until four years later, when Chaussier took up the anatomy of the diploic veins anew in his book (*Exposition sommaire de la Structure et des différents Parties de l'Encéphale ou Cerveau*). This author gives an admirable description of the vessels, and accompanies it with a single clear explanatory plate. But he makes no mention of Dupuytren, and, indeed, so far is he from crediting Dupuytren with their discovery that he states on p. 18 of his preface that the diploic veins "have been discovered by M. Fleury, prosector of the École de Médecine, and now doctor and surgeon-in-chief of the Hospital of Clermon-Ferrant." These bibliographical points are interesting and rather confusing, and our confusion is increased when we find that anatomical text-books to-day almost all attribute the veins to yet another person, the distinguished French anatomist Gilbert Breschet. It was not until 1819 that anything on these veins came from Breschet's pen, the first of a series of three papers, each of increasing importance, and culminating

in the magnificent volume of studies which began to appear in 1830 and was never completed. The solution of the problem as to priority is to be found in a paper by Raciborski, " Histoire des Découvertes relatives au Système Veineux," 1836, from which we gather that Chaussier was indeed correct when he attributed the discovery of the veins to Fleury. Raciborski refers the reader to a letter written by Fleury to the *Gazette Médicale* in 1836 in which Fleury states that he found the diploic veins by chance whilst preparing skulls for the museum. He removed the outer table to exhibit the diploë, but going a little deeper than usual came across the venous channels. He told this fact to his intimate friend Dupuytren, who made it the subject of a memoir read to the Société de l'École de Médecine, and later included it in the thesis of 1803 to which reference has already been made. Raciborski remarks, " If we regard Dupuytren as the first of the authors who have spoken of these venous channels in the bone, we ought always to render justice to M. Fleury and regard him as the author of this discovery." He refers also to the enmity

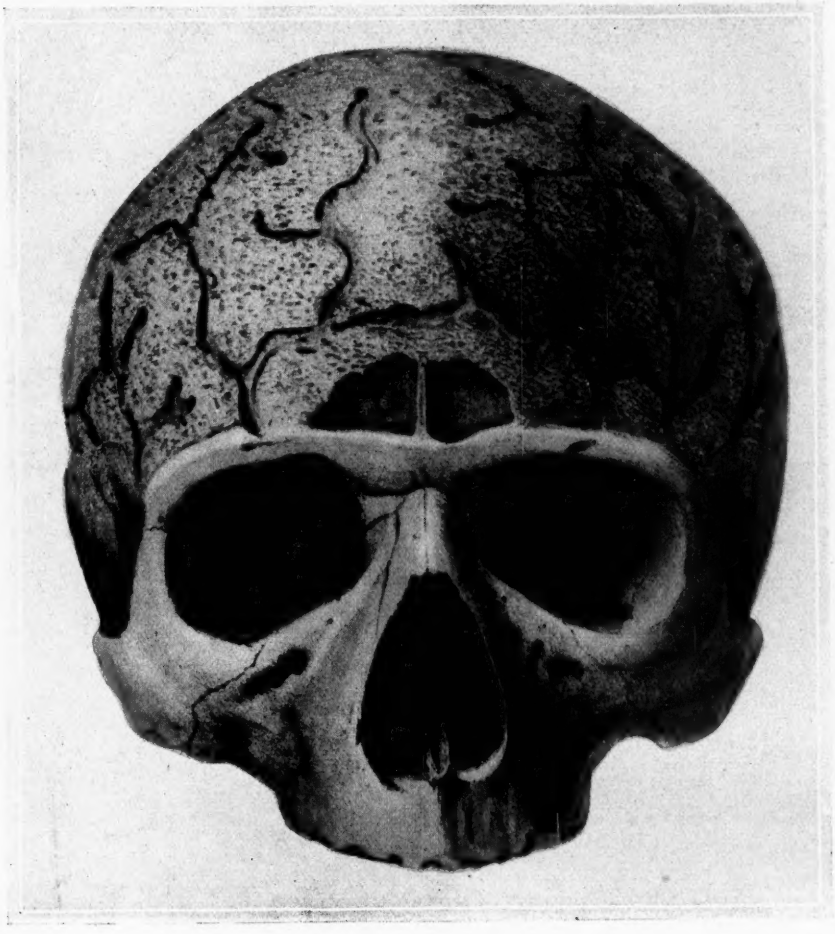

FIG. 47.—The frontal diploic veins. Note their emergence at the well-known emissary foramen in the supra-orbital margin (*Breschet*).

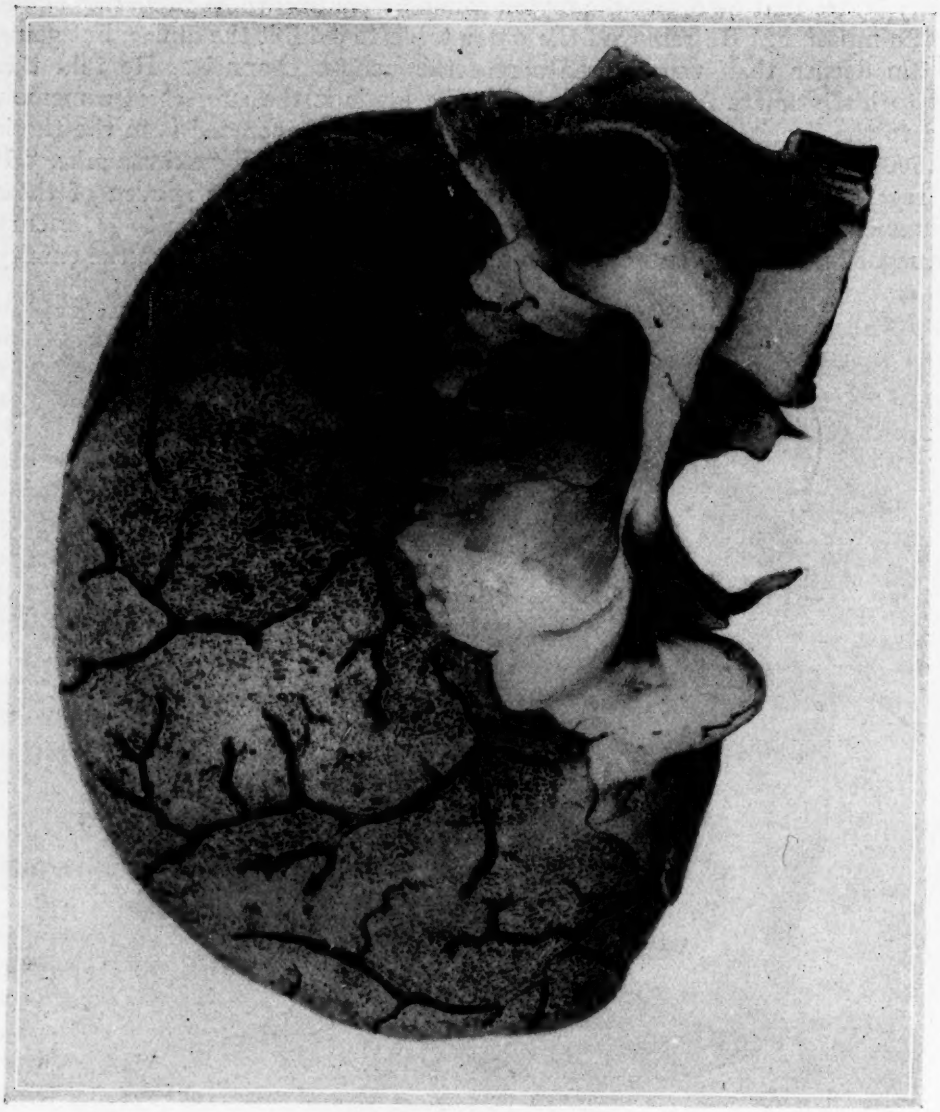

FIG. 48.—Lateral view to show the anterior and posterior temporal diploic veins and their connections (*Breschet*).

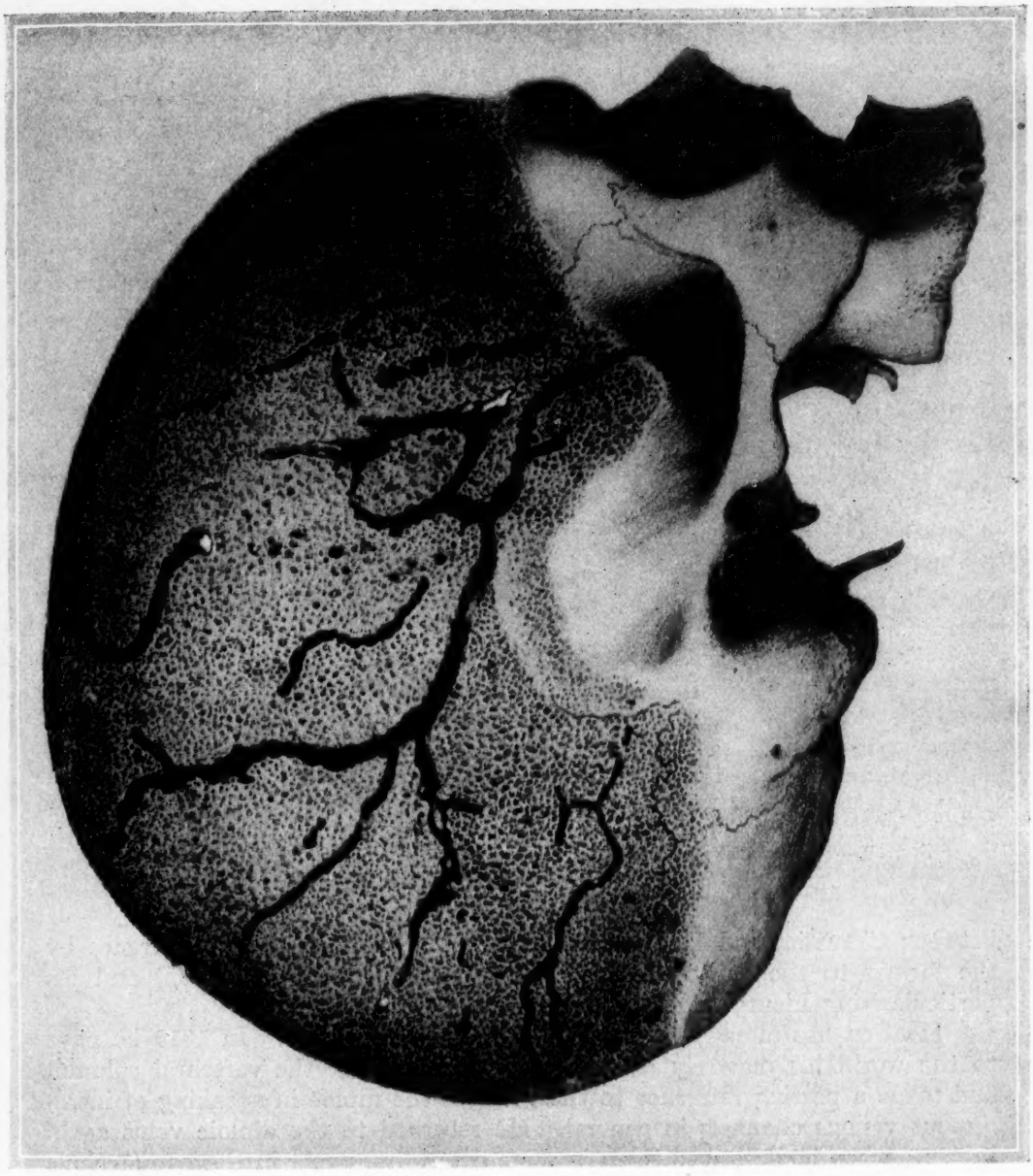

FIG. 49.—To show variations and communications with the meningeal veins (*Breschet*).

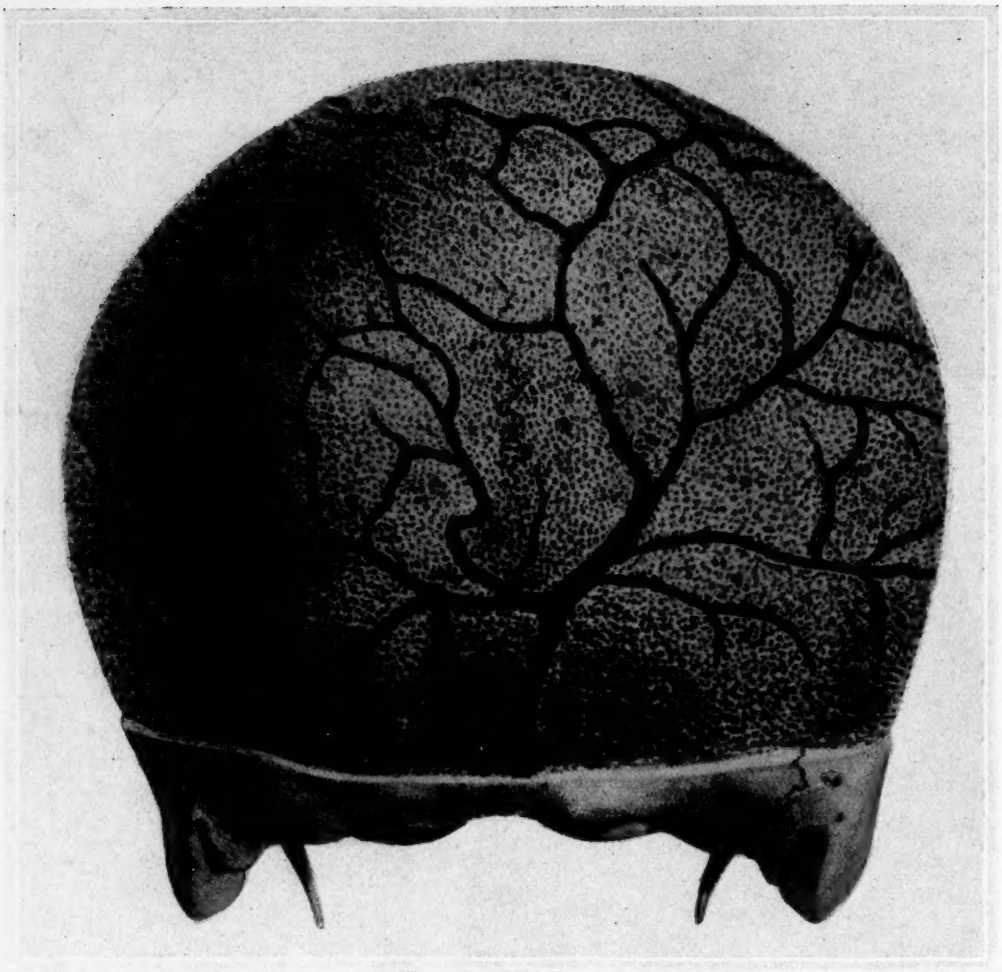

FIG. 50.—To show the dispositions of the occipital diploic veins, communicating with the posterior temporals or parietal veins (*Breschet*).

between Chaussier and Dupuytren, whence the omission of any reference by the former to the early paper on the "canaux veineux des os," and his particularizing Fleury, in his preface, as the real discoverer.

Mention has already been made of Breschet's papers. In 1819 he gave to the world his masterly exposition of the veins of the vertebral column, and made a passing reference to the veins of the diploë in speaking of intra-osseous venous channels in general. He returned to the diploic veins again in 1826, when he described them in some detail in a paper in German ("Nova Acta Physico-Medica, Naturæ Curiosorium," Bonn). He describes their development from half-canals at the centres of ossification of the principal bones (this, in passing, the present writers have confirmed). He also describes the minute pitting of the walls of these canals, proving that they are fed by innumerable venules from the meshes of the diploë.

Breschet's real claim, in popular estimation, to association with the diploic vessels, rests without question on the publication in 1830 of a superb volume, *Recherches Anatomiques, Physiologiques, et Pathologiques sur les Canaux Veineux des Os.* This beautifully illustrated work was an ambitious undertaking, and Breschet did not live to see it completed. It is an ironical fact that although there are twelve chromolithographic studies of the veins (four of which we are able to reproduce, by courtesy of the Librarian of the Royal College of Surgeons), there is no description of them in the text, no doubt owing to the unfinished state of the monograph. But judging from the plates it is evident that Breschet's studies were very complete, and had been carried further since his German publication. His account of the spinal veins has never been surpassed, and remains the standard description to this day. No doubt the diploic veins would in time have been equally described; in any case the beauty of the plates and their self-explanatory clearness has compensated for lack of text, and caused his name to be associated by a later age with this system of calvarial veins. In this posterity has unwittingly given him greater credit for originality than was his due, but knowledge advances step by step, and the final embellishment may be worth more than the bare outline of a first observation.

Forty years later Trollard, in a *Thèse de Paris* ("Recherches sur l'Anatomie du Système Veineux du Crane et de l'Encéphale"), enters into a new description of the veins. He dissociates himself from the classical descriptions of Chaussier and Breschet, believing that the diploë is a spongy tissue with areolæ full of blood. The walls, according to him, are absorbed as age advances, and thus irregular channels are formed, but no definite or constant venous trunks. The earlier writers had believed that the vessels which they could easily trace with the naked eye arose from small radicles. This belief Trollard also combats, setting up in its place the idea that the veins start in blunt lacunæ, acquiring at once their full width. This point is worth mentioning, because on rough dissection the veins do indeed often appear to commence in this wise. We shall see, however, that this conception is incorrect. All veins save the largest trunks do notoriously vary greatly, and, so far as concerns this point alone, Trollard may have had some justification for his views. But modern injections and X rays prove that he was fundamentally wrong.

Another French monograph, one by E. Boismorreau, deserves something more, perhaps, than passing reference. In 1904 he published his inaugural thesis, "Contribution à l'Étude de la Vascularisation du Diploë," and gives a very sober and well-documented description of the diploë and its vessels, confirming the views of the earlier writers as against those of Trollard.

One last paper calls for comment. Elsberg and Schwartz have described the radiographical appearances of these veins with special reference to meningeal endotheliomas, and have given them a certain significance. Our own observations show that the veins may be well seen in perfectly healthy persons, whilst in a large series of tumour cases only occasionally was it possible to establish any notable increase in the size or field of distribution of the veins.

THE NORMAL VASCULARIZATION OF THE BONES OF THE CRANIAL VAULT.

The worker eager for information on the normal means of vascularization of the cranial vault will find very little to help him in readily accessible literature. It is evident that the flat bones of the skull, having no epiphyses, and a natural history of growth and function which differs widely from the long bones of the limbs, will have a blood-supply differing quite distinctly. In the long bones there is a system of nutrient arteries, one (or more) of which enters the shaft and divides to run to the opposite ends of the medullary cavity, where it anastomoses with the metaphysial sets. These nutrient arteries, despite the somewhat large openings in the cortex of the bone, are usually surprisingly small when studied by opaque injection and X rays. The calvarium, on the other hand, presents a different picture. In the place of the single nutrient foramen there are a number of small openings, limited to well-defined areas, the sites of which differ materially on the inner and outer surfaces. The outer table is generally smooth and very faintly pitted, but no openings large enough to admit a needle point will be found except in the areas of muscular attachment, as in the anterior part of the temporal fossa, and particularly in the occipital region below the superior curved line, where a variably large number will be found. These give entrance to branches of the muscular arteries which pierce the bone for its supply, fine vessels with a short course, making up in number what they lack in size. The foramina equally give exit to diploic veins. It is a commonplace experience of cerebellar surgical approach to meet with free venous oozing below the fringe of muscular attachment as the muscles are swept off the occipital bone. The divided vessels are apt to be loosely referred to by the operator as efferents of the lateral sinuses and torcula. More often they are not true emissaries, that is, freely perforating vessels, but are diploic efferents. This is clearly demonstrated by the behaviour of the troublesome bleeding points often encountered about the external occipital crest, for bleeding from them is easily stopped with wax, and when the bone is rongeured away the dural surface beneath is smooth and dry.

Apart from diploic connectors of this kind there are no noteworthy openings on the cranial vault save those of the well-known symmetrical emissaries, the mastoid, parietal, and so forth. Careful study of these latter foramina will frequently show that as they pierce the bone they are joined by diploic veins, a small perforation or two being discoverable in their smooth walls.

The arrangement of nutrient vessels is quite different on the inner surface of the skull, which, apart from the irregularities due to convolutional marking, presents the characteristic meningeal vessel grooves. On this surface again we find the multiple foramina which we saw at certain points on the external table, and once more they are strictly limited in their distribution; but now they occur only in the meningeal grooves and in and about the grooves of the great dural venous sinuses, a fact which gives us a clear lead as to the function of the meningeal vessels.

The Meningeal Arteries.—The meningeal arteries, and particularly the middle, are so hallowed by association with a clinical syndrome, that the reason for their presence may often escape observation. We may well ask ourselves why a fibrous membrane which should require no more vascularization for its own needs than the fascia lata of the thigh should be so richly supplied with vessels. For the dura is indeed very freely supplied with vessels—vessels so small and so easily controlled at operation that one is apt to be misled as to their number. We must confess that, with preconceived views formed at operation, injected specimens of dura came as a surprise;

FIG. 51.—Radiograms of calvarium of dry skull. Diploic vessels in blue, meningeals in red.

many fine branches will be seen ramifying up to and beyond the sagittal sinus. The injections confirm the absence of any special meningeal arterial supply to the Pacchionian bodies. But from our point of view at the moment, the small puffs of lipiodol seen along the vessel and at the ends of the twigs of the arterial tree are of more importance. These represent the points at which these fine vessels were entering the bone as nutrients. When the artery, on such a specimen, is washed out with water, small pools collect at many points for the same reason. Conversely, when water

is forced into any channel in the sawn edge of a removed calvarium at post-mortem the inner table is seen to 'weep' in like manner. It is quite evident, then, that the meningeal vessels are not destined for the supply of that membrane alone, but rather for the overlying bone, of which the outer layer of the dura has long been known as the inner periosteum. At the point where the middle meningeal vessels tunnel the greater wing of the sphenoid, opportunity is taken for the interchange of vessels with the interior of the bone, venous connections being made here in particular,

FIG. 52.—Lipiodol injection of the meningeal vessels in the bone.

though anastomoses occur besides at all points along the course of the vessels.

Meningeal Grooves.—It will be admitted by most who have worked at the subject that Wood Jones was correct when he re-established the old teaching and assigned to the veins the chief part in the formation of the meningeal grooves. So often at post-mortem one finds a deeply-cut groove running up to the parasagittal sinus and Pacchionian markings, whilst the artery supposed to have caused the bony track is seen tapering away until it is so thin above as to have been quite inadequate to occupy the wide furrow. It is a fact that the middle meningeal vessels, and their grooves, make their way up to the Pacchionian bodies, essentially venous structures. There is a capillary system at the origin of these veins, for on section in the living there

is not that severe reflux bleeding that would occur if they connected directly with the sinus.

The Vascularization of the Calvarium.—In summary it may be said that the bones of the vault are supplied with blood by branches of the meningeal vessels, and particularly the middle meningeal; that these branches penetrate the inner table and are generally of small size. Reinforcement is effected by arteries which enter the outer table, particularly in the areas of muscular attachment. But the larger channels in the bone are, as we shall see, essentially venous, an anatomical corroboration of another operative observation—that bleeding from the cut or nibbled calvarium is predominatingly venous in type. The vascularization of the cranial bones may be regarded as a seepage from very numerous vessels. It seems probable that this is not a unique method of circulation in bone, for the supply of the ends of the long bones and the compact tissue is of a rather similar type. In the cranium it would appear that the meningeal arteries replace the long nutrient arteries of the limbs, the extra-osseous course of the former being an exigence of different bony function and structure. Now it is a well-accepted fact that an osteoplastic flap may be lifted without fear of necrosis of the bone, so long as it remains attached to the temporal muscles: adhesion to the overlying skin is not essential. There is no question that at times replaced bone-flaps must approach the category of free grafts; and it is equally a fact that porosis of flaps does at times occur, though rarely. These bone segments are free of the dura when the bone is reflected and the fine vessels entering from this side are torn off. This deprivation is, as has been said, not a serious matter, so that whilst one may truly lay it down that the meningeal vessels are largely for the use of bone rather than of the fibrous matrix in which they lie, they are not in themselves essential for the continued life of that bone. Not only will the bone survive, but it is at times re-united by bridges of new bone. Union may be expected more often in the young, though we have seen it in older people; there seems to be no rule on this point. A flap a year old in a man of 30 was recently re-turned and fibrous union only was found; in another, re-opened after nine months (male, age 42), there was firm bony union along one border but not elsewhere; in yet a third (male, age 30) the flap six years later was found to have re-united in almost all of its circumference, though a groove marked the position of the original section.

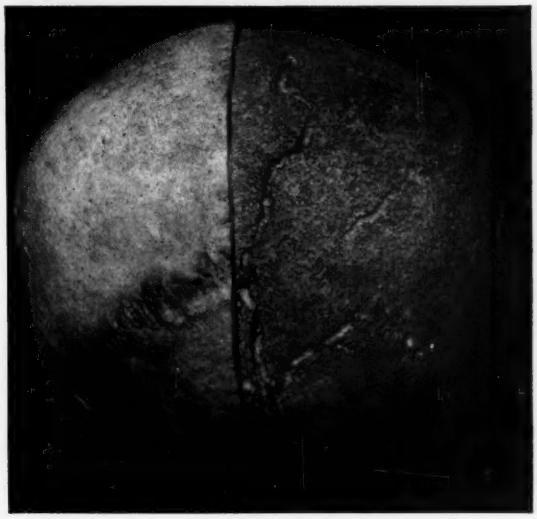

FIG. 53.—The occipital veins of the diploe. (From a specimen in the Museum of the Royal College of Surgeons, Edinburgh. Note the depth from the surface.)

THE ARRANGEMENT OF THE DIPLOIC VEINS.

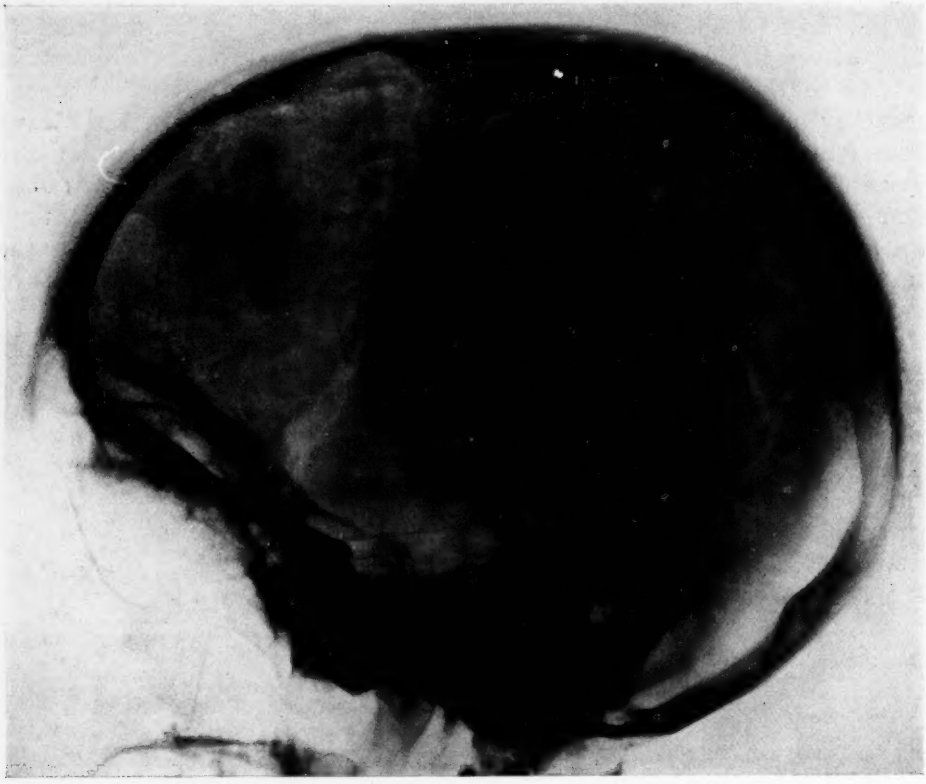

FIG. 54.—Diploic veins from a normal man of 55.

Breschet's own figures can scarcely be bettered, so we have chosen to illustrate this note with four of them (*Figs.* 47–50). They are so clear that little explanation is needed. As with venous channels elsewhere, considerable variation in size and position is to be expected. They lie nearer the inner than the outer table, and whilst each of the bones of the vault seems to have a set particularly of its own, connecting branches cross the sutures to unite the various groups. The veins, as seen in skiagrams of the living, group themselves into three main sets, frontal, parietal, and occipital. A V-shaped arrangement with the apex downwards is very characteristic (*see Figs.* 47, 48, 50), especially in the frontal region. The exit of these frontal veins by a small foramen in or about the supra-orbital notch is clearly seen in most dried skulls. The occipital set drain into large channels, one on either side of the mid-line, converging towards the base. The much larger parietal set includes the anterior and posterior temporals described by Breschet and others. Of the two, the posterior group are those most commonly seen in X-ray photographs of the living, and connections with the occipital diploic vessels are common, whilst the anterior temporal diploics are hard to see, for confusion arises between them and the grooves of the meningeal vessels,

with which they are closely connected. *Fig.* 50, the skull of an old man, shows actual defects along the course of this vein. Examination of the meningeal grooves in the inner table of the aged reveals, not the fine pitting of younger skulls, but many places where the floor of the groove is missing to a greater or lesser extent. Here the two systems become confluent.

It is important to recognize that this anastomosis takes place between the diploic channels and both the anterior and posterior divisions of the middle meningeal, for on the skiagrams of living skulls it is not uncommon to see the meningeal grooves run back and up, to be lost towards their termination in a typically diploic constellation.

We have confirmed the course and distribution of the diploic veins by the study of X-ray photographs both of the living and of dry skulls. Three attempts at injection on the cadaver with Ferguson's mass did not give the results expected. In the dry skulls the specimens used were either calvaria which had been separated from the remainder of the skull by a horizontal cut, or lateral halves of skulls which had been divided in the sagittal plane. By using these it was possible to avoid the images of the veins of the two sides being superimposed on the film. It had, however, the disadvantage, in the specimens of the calvaria, that only the superior portions of the veins could be seen.

When the X-ray films of the dry skulls (*Fig.* 51) were examined and compared with the dural aspects of the skulls themselves, it was found that not only were the grooves for the meningeal vessels shown on the film, but also other impressions obviously made by other blood-vessels. These were the diploic veins. It is not a difficult matter to distinguish the diploic from the meningeal channels without examining the inner aspect of the skull, as the former have certain characteristics: (1) While the general course of the meningeal vessels is upwards and backwards from the region of the small wing of the sphenoid, the diploic veins often run at right angles to this direction (especially in the posterior parietal region). (2) The origin of the larger diploic veins tends to be blunt. (3) There is a curious star-like branching and anastomosing of veins, which we term the 'spider' (*Figs.* 56–59). This is usually best seen in the parietal region, but may occur elsewhere. (4) The Y-like branching of the veins, commonly with short thick limbs, may be clearly made out.

When skiagrams are taken of the living skull, the results are complicated by the superimposition of the grooves of one side upon those of the opposite side, when seen in a film. The diploic markings, however, have the same characteristics as seen in the dry skull, which renders it comparatively easy with practice to distinguish the diploic veins from the meningeal vessels. The edges of the diploic grooves are quite sharply defined, but frequently show a recognizable crenation.

In the film of the dry skull, although it was quite simple to trace out the course of the main diploic vessels, it was difficult to differentiate the finer grooves by simple comparison of the skull and the film. To overcome this difficulty the following technique was adopted. On a matt print of a film all the larger meningeal grooves, as observed on the inner surface of the skull, were painted in red, and then by comparing the skull and the film all the larger

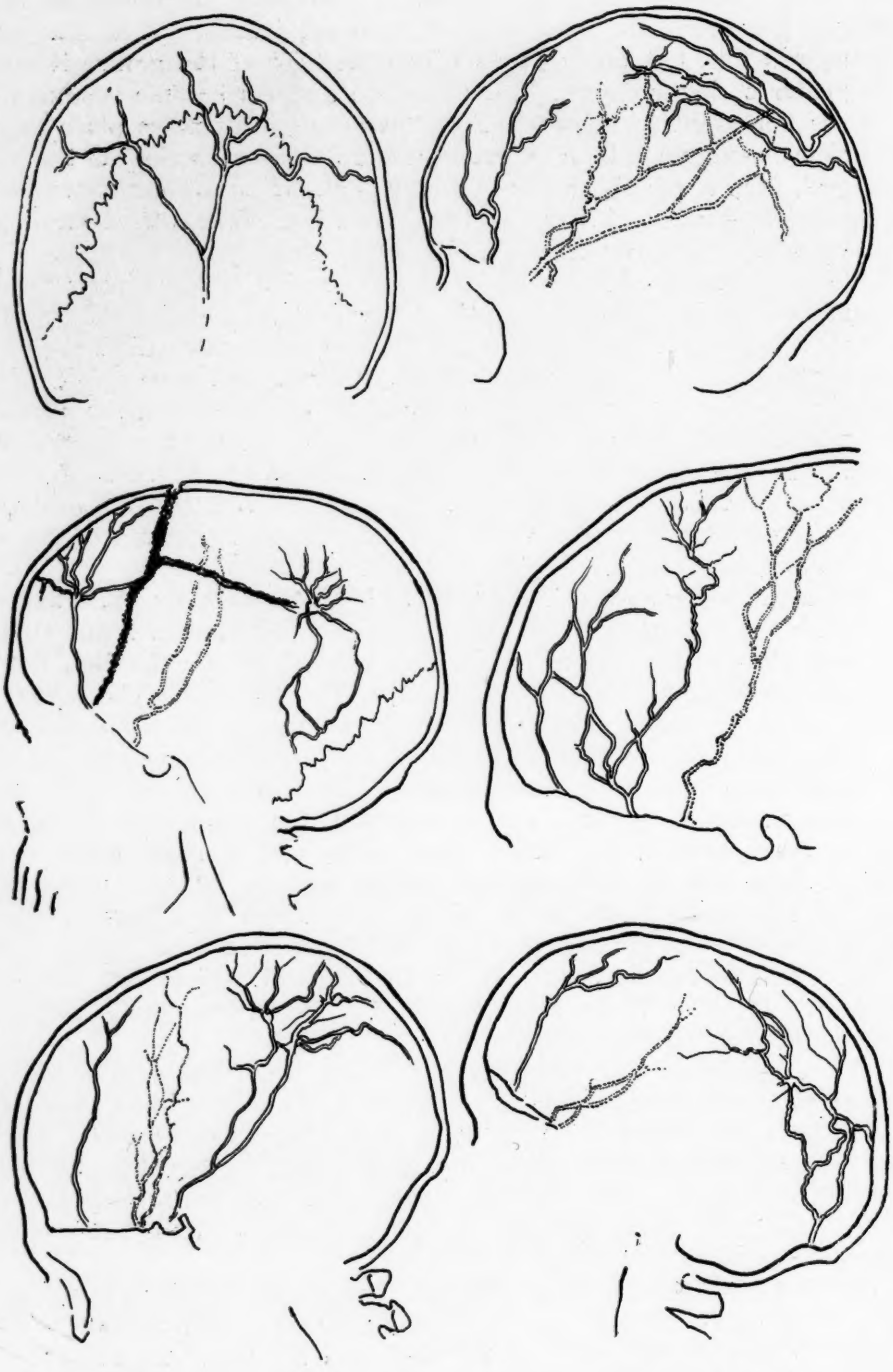

FIG. 55.—Drawing from X-ray photographs of variations in the diploic veins (continuous lines) and meningeal vessels (dotted lines). In the third there is a fracture; the horizontal limb was reported as ending in a "stellate crack."

DIPLOIC VEINS SHOWING THE 'PARIETAL SPIDER'.

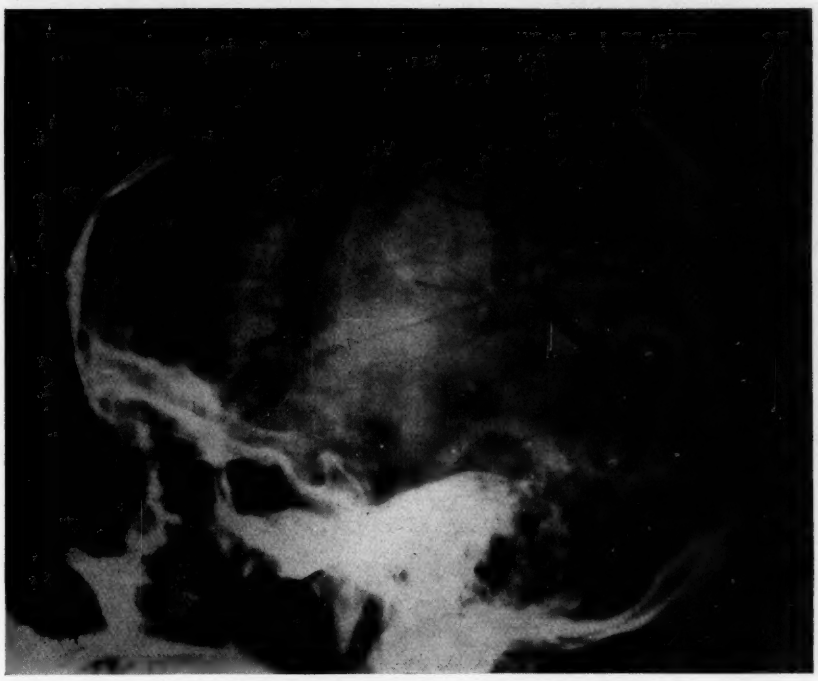

FIG. 56.—A male, age 24.

FIG. 57.—A male, age 28.

DIPLOIC VEINS SHOWING THE 'PARIETAL SPIDER'—*continued.*

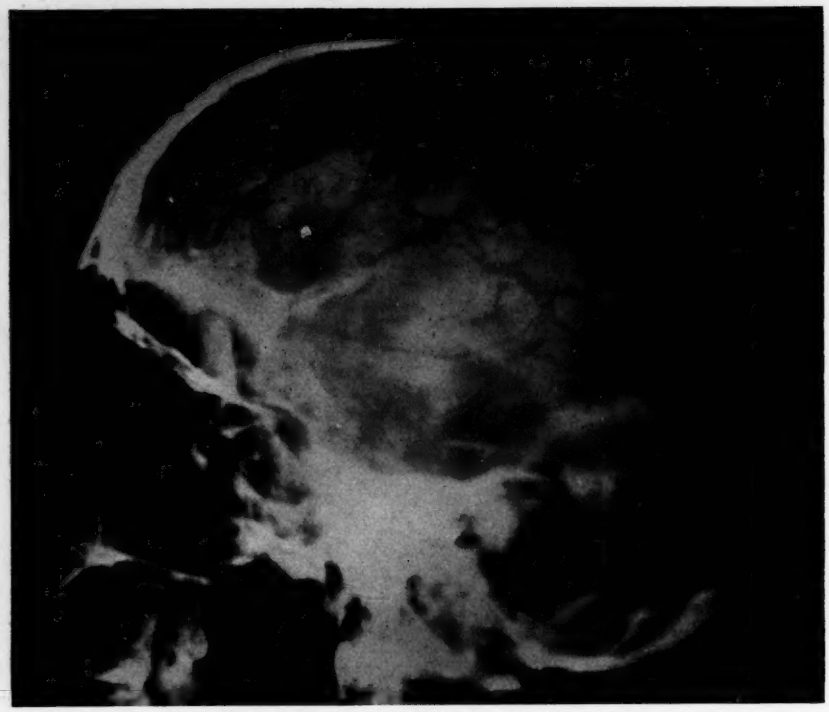

FIG. 58.—A male, age 48.

FIG. 59.—A female, age 50.

diploic grooves were painted in blue. Then by a careful examination of the film, which showed more details than the print, it was possible to differentiate and paint in many of the smaller vessels. *Fig.* 51 shows a print dealt with in this way, and gives a very clear idea of the distribution of the diploic veins. The results obtained by the methods described above generally confirm the findings of the older observers. The frontal, posterior, temporal, and occipital veins were easily traced, and it was found that the posterior parietal and the occipital constantly and freely anastomosed with each other in the parietal region, forming the 'spider.' The frontal diploic veins were more isolated, and connections could only rarely be traced between them and

FIG. 60.—Diploic veins in a case of adult hydrocephalus of long standing showing the lakes of confluence.

the other veins. It will be noticed that no mention has been made of the anterior temporal vein. This is due to the fact that it was not seen in the dry skulls and cannot often be unequivocally distinguished in the living. Its place frequently appears to be taken by a vessel grooving the inner aspect of the skull in front of the middle meningeal grooves, which frequently originates in a depression near to the great longitudinal sinus; it ends in the sphenoparietal sinus. We are of the opinion that this vein may be the anterior temporal diploic vein placed more deeply in the dura than the other diploic vessels, which are placed more superficially in the bone.

Its fate seems therefore to be bound up with that of the anterior division of the middle meningeal vessels. *Fig.* 60 is from an old-standing hydrocephalus (female, age 24). The sharp-cut groove for the middle meningeal vessels is replaced by a chain of small pools of blood. This figure may be most usefully compared with Breschet's figure (*Fig.* 49). (This possible interchange has been noticed by Coen, and is referred to in a paper by Thompson, which we have seen only after the above was written). The very deep markings of the meningeal grooves figured by Elsberg and Schwartz may have a similar genesis.

CLINICAL APPLICATIONS.

In surgical practice the veins of the diploë will most often cause confusion in X-ray cases of head injury (*Fig.* 61). It was, indeed, the report of a radiographer that extensive stellate fractures were present in the parietal region of two patients who had had no injury at all, that drew the attention of one of us to the problem of the calvarial vessels. Generally a fracture line is easy to

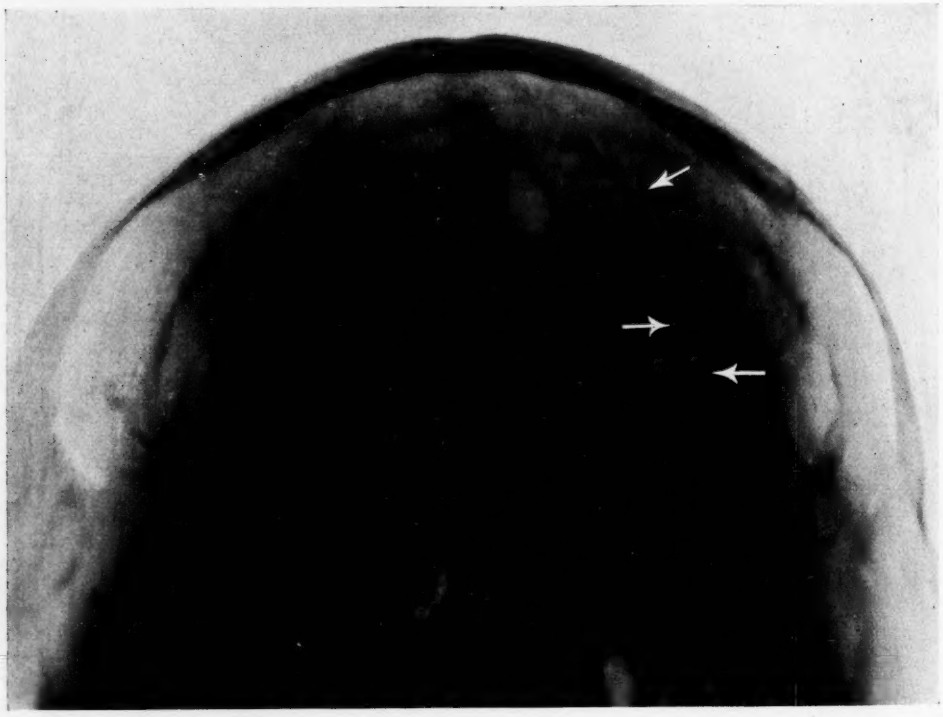

FIG. 61.—X-ray showing diploic veins on the right, and a fracture cutting the left orbital margin and simulating a diploic vein. The line of fracture is shown by the arrows.

distinguish owing to its straightness, its clear-cut margins, and its extent. The great safeguard is an adequate knowledge of the normal appearances of the veins, their position and direction, their tendency to anastomose and form 'spiders,' stars, and Y-junctions. We have found clinically that the most difficult tracks to assess are those parallel with the contour of the skull high in the parietal region. It must be remembered that the veins, like

fractures, often run across suture lines, but anyone familiar with them will hardly fail to distinguish them from injuries. A mistake in a case involving a civil action at law, as in the now common motor accident, may be costly. Further, it is possible that deep venous erosions may assist in determining the line of fracture, as Elliot Smith showed years ago and as Thompson again suggests.

A dilatation of the veins in the diploë might be thought to be a likely concomitant of intracranial tumour, with its pressure on the venous side. We have referred to the serious venous oozing met with at certain times and in certain places, and emphasized that the bleeding is commonly from diploic efferents as well as from perforating emissaries. Now it is quite true that the eye skilled in the detection of the diploic channels in skiagrams of the living may frequently note here an unusual clarity in the vessels' lines, there an uncommon pool of blood. Yet the fact remains that the most beautiful examples of diploic veins that we have seen have been discovered by chance in skiagrams of non-tumour cases. The observer would therefore be very wrong if he concluded that because the veins were very distinct a tumour was present in the skull. The age factor has been remarked on. In children with their sparse diploë the veins are small and hard to define ; as age advances they become clearer. When the skull is thinned by expansion in hydrocephalus above infancy the veins are again ill-seen, save that around the great meningeal collecting trunks diploic markings become more evident (*Fig.* 60). But it is impossible to lay down a rule that shall be true of every case ; some will demonstrate one point and not another. There seems to be some ground for the views of older writers, that the diploic system acts as an additional reservoir for the blood circulating within the cranium and might act as an escape or balancing mechanism when that current is disturbed. Operative experiences with tumour cases support this belief.

In meningioma involving the overlying bone there is no question as to the increased diploic bleeding, clearly localized as to its source by the uniform failure of ligature of the external carotid artery to abate it. It is evident that the time factor must be very important, and that if diploic irrigation is to be increased in a tumour-bearer a considerable period must elapse before dilatation recognizable on an X-ray film can occur. We can only repeat that inferences drawn from the radiological appearances of these veins without reference to other more important clinical factors will almost certainly be faulty.

One last word, and that on infection. Acute pyogenic osteomyelitis is rare in the vault owing to the absence of epiphysial lines. On the other hand, the venous channels allow of easy spread from one area to another, and this explains the patchy distribution of calvarial infection when it does occur. A case recently observed will serve as illustration. A young man began with a furuncle on the nose which rapidly caused œdema and cellulitis in the orbit. After a stormy convalescence a deep temporal abscess formed. Later patches of osteomyelitis developed in the frontal and high in the parietal region. Infection could easily find its way by means of a septic thombophlebitis originating in the deep veins of the temporal fossa and spreading by way of the vessels, which enter and leave the bone in this region, with septic permeation and osteomyelitis at a distance.

SUMMARY.

The discoveries of our forefathers have a habit of attaining a new importance many decades later. This is exemplified by the veins of the cranial diploë, which refinements of X-ray technique now render clearly visible in most radiograms of the living skull. These veins, which occupy very definite channels in the substance of the bone, may be easily mistaken by the unwary for fractures. Generalized enlargement, detectable by radiography, is not the rule in cerebral tumour, and beautiful examples may be met with in normal persons, especially in the later decades of life. Unusual clarity and breadth of the middle meningeal grooves is more often met with in cases of intracranial tumour. In these cases the anterior temporal diploic veins add their quota to the impress of the meningeal vessels. The meningeal vessels, both arteries and veins, are destined to a considerable extent to supply the bone.

Figs. 47 to 50 are photographs from Breschet's book made by kind permission of Mr. V. G. Plarr, Librarian to the Royal College of Surgeons. *Figs.* 51, 55, and 60 are by Miss Davison. We are indebted to Mr. Greig for *Fig.* 53. We have to acknowledge also the interest which Mr. Higgins, Radiologist to the Salford Royal Hospital, has taken in the work, and are indebted to Dr. Morris, of the same department, for the prints of *Figs.* 56–59. Professor Stopford has been most kind (as ever) in allowing the free use of material in the Department of Anatomy.

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PSEUDOCOXALGIA: CLINICAL EVIDENCE, X-RAY APPEARANCE, ETIOLOGY, AND TREATMENT: WITH A REVIEW OF 25 CASES.

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SYMPTOMS AND PHYSICAL SIGNS.

Frequently the symptoms and signs of pseudocoxalgia are so indefinite at first that the disease is unnoticed in the earlier stages.

The most constant symptom during the onset of the disease is a limp: there is a tendency to drag the affected limb, the foot is not lifted freely from the ground, but is swung round in an everted position with each step. The limp is not painful and is frequently intermittent; for two or three weeks at a time it may not be present. After from six to eighteen months, when the disease has become more fully established, the limp frequently disappears, but if it persists it may change its character and resemble the 'pain' limp found in infective arthritis of the hip-joint. Pain is said to be absent in nearly half the cases, but it will be seen that it was present in the majority of my patients at their first examination. It may be slight and localized to the hip, or referred to the knee, while in a few cases it is extremely severe.

Some degree of muscle-spasm can usually be found during the early stages of the disease. As a rule it only lasts for a short time, but may recur, and often persists longer than the pain and tenderness. This will result in a painless limp, the limb being fixed in an adducted and slightly flexed position, in contrast to the abduction found in early tuberculosis.

The amount of movement possible in the joint depends largely on the degree of muscular spasm, and will therefore vary considerably. Frequently the movements are nearly perfect throughout the disease, and even if limited at first they soon improve; but usually there is some limitation of abduction and internal rotation, which often persists in these two directions throughout life. Sometimes the hip may be nearly completely fixed, usually in a position of adduction and slight flexion.

Muscular atrophy may be either hardly noticeable or well marked. The localized thickening of the soft tissues so frequently felt in tuberculosis of the hip-joint is not found in pseudocoxalgia. The great trochanter of the femur is prominent and projects laterally. Observers differ as to whether it is found at a higher level than normal; in my own cases no such alteration was noticed. Abscess formation never occurs in this condition.

Osteo-arthritic changes take place in a certain number of cases at a late stage, and result in pain, stiffness, and further limitation of movement.

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* A paper read before the Sheffield Medico-Chirurgical Society, October, 1927.

From a consideration of the clinical evidence of pseudocoxalgia it will be clear that it is often extremely difficult to distinguish the condition from epiphysial coxa vara, tuberculosis, and other diseases. In fact in some cases it is quite impossible to do so with certainty by ordinary physical examination, but as the radiographic appearances precede the clinical signs the diagnosis can usually be made by the X rays.

SKIAGRAPHIC APPEARANCES.

Changes in the Head of the Femur.—In a normal child the head of the femur is cartilage with a bony nucleus in its midst which throws a shadow in the skiagram. The extent of the cartilage can be estimated by noticing the size of the clear space between the nucleus and the acetabulum.

1. *Stage of Flattening.*—At first there is an increase in the density and a diminution in the size of the bony nucleus, making the head of the bone appear flattened. The size of the epiphysis remains unchanged, but the joint interval appears larger as less space is occupied by the bony shadow (*Fig.* 62, p. 95).

2. *Stage of Fragmentation.*—The bony nucleus next begins to show irregular calcification; parts show increased density, while others undergo rarefaction. This process continues until the bony nucleus appears broken up into fragments—areas where lime salts are markedly increased in amount, with intervals of decreased calcification between them (*Fig.* 63, p. 95). At this stage, being deprived of its bony support, the cartilage begins to give way; it loses its spherical shape and becomes spread out laterally. The process of fragmentation may be very little marked, or it may continue until the head of the bone has scarcely any bony nucleus left.

3. *Stage of Repair.*—It should be remembered that the cartilaginous envelope is deformed; it is flattened and squashed out laterally, so that it envelops the circumference of the acetabulum, and when the bony nucleus re-forms, its shape is limited by the boundaries of its cartilaginous surroundings. The fragments begin to coalesce; then the density of the whole epiphysis gradually diminishes until it becomes uniform and equal to that of the opposite side. Eventually it increases in size until it nearly fills its flattened cartilaginous envelope (*Figs.* 65–70, p. 95).

4. *Stage of Moulding.*—The re-formed femoral head undergoes moulding until adult life. It is said that one of two types of head results, either (*a*) a globular, round head, or (*b*) an oval head. Legg[2] calls these the 'mushroom' and 'cap' types respectively, and it is generally thought that these two types are quite distinct, and that the function of the former is ultimately superior to that of the latter. In my experience it is often very difficult to know in which category to place many cases, for the line dividing the two types is frequently poorly defined. With regard to the ultimate degree of function, all that can be said is that the more the shape of the head differs from the normal the worse it will be. This will become more noticeable in later life, as in cases with deformed heads osteo-arthritic changes are likely to occur.

Changes in the Neck of the Femur.—The contour of the neck alters, its upper part becomes broader and its upper angle rounded off, while later on there is shortening of the neck as a whole, producing an apparent coxa vara.

During the beginning of this stage an ill-defined zone of change in calcification produces a sponge-like area in the upper part of the neck. This metaphysial change is early and constant (*Fig.* 71, p. 96). Later a regular pattern-like appearance is sometimes noticed; there are zones of increased calcification arranged as pockets opening towards the epiphysial line, each pocket enclosing a corresponding area of rarefaction. This phenomenon is said to be associated with fragmentation, and to disappear early during the period of healing, but in the few instances where I have noticed the appearance it has been quite a late occurrence.

Changes in the Acetabulum.—Opinions differ as to whether any changes are constantly found in the acetabulum; and whether these are primary if they do occur, or only late hypertrophic changes secondary to those in the head. Perthes[3] says that the acetabulum only shows changes in advanced stages, while Sundt[4] considers that the acetabulum is normal late in the disease. Platt,[5] however, thinks that changes "can be seen at every stage, and that they should be considered partly as the adaptation of the cavity to the altered lines of pressure through the deformed head, and partly as of the same nature as the transformation undergone by the epiphysial nucleus and thus truly specific."

ETIOLOGY.

Age.—Pseudocoxalgia occurs most often between the ages of 5 and 10.

Frequency.—Calvé[6] found 1 case in every 50 patients with hip disease. Gauvain[7] reports an incidence of 1 in 24, while during the last five years the present writer has seen 25 cases among 360 patients with alleged tuberculosis of the hip-joint, a proportion of more than 1 in 15.

Sex.—Boys are more commonly affected than girls; most authorities consider the proportion to be about 4 to 1. Platt,[5] however, in his 23 cases, found 12 boys and 11 girls. In the present series of 25 cases there were 18 boys and 8 girls.

Bilateral Cases.—These are comparatively few. Legg[8] found 2 in 55 cases; Perthes,[3] 2 in 23; Platt,[5] 2 in 23; Sundt,[4] 7 in 75; Pattison, 5 in 25.

Heredity.—Although a few cases have been reported in which the disease occurred in the same family, heredity does not seem to be a factor in the incidence of pseudocoxalgia. Neither syphilis nor rickets seems to be associated with the condition.

When investigating the cause of a non-fatal, chronic disease such as pseudocoxalgia, we are handicapped for lack of material for pathological examination. A number of surgeons have, however, operated on patients with this condition, either to determine the nature of the disease or in an attempt to remove the focus and so hasten the cure. It may be well to consider what has been found by exploration before examining the theories advanced to explain the pathogenesis of the condition.

The synovial membrane has sometimes been normal in appearance, but in a few cases it was thickened, showing evidence of mild synovitis; in these cases the joint-cavity was filled with a slightly turbid and straw-coloured fluid.[9] No bacterial cultures have ever been obtained from the synovial fluid; nor has inoculation of guinea-pigs with the fluid shown evidence of bacterial infection.

Perthes[3] removed a small piece of the head of the femur. The interior of the epiphysis showed replacement of the cancellous tissue by invading buds of cartilage; he considered the condition to be non-inflammatory. Legg[8] scraped out a large area of rarefaction in the neck of the femur and grew staphylococci from the débris so obtained. These staphylococci were very scanty and may have been due to contamination. Kidner[10] and McWhorter[11] also obtained isolated colonies of staphylococci from the granulation tissue found in necrotic areas in the metaphysial region. Phemister[12] removed part of the epiphysis after curetting the interior. He found that the joint-cavity showed evidence of acute synovitis, although the articular surface of the deformed head retained its normal sheen. He obtained no microbic growth from the material, but observed changes which he considered typical of an old infective lesion of the bone, and probably of pyogenic origin.

None of the many theories which have been brought forward to account for the pathogenesis of pseudocoxalgia is alone adequate to explain the condition. A few of the most reasonable theories will be briefly considered.

The Infective Theory.—The exponents of this consider that pseudocoxalgia is produced by infective agents of low virulence. It has many supporters and has been ably upheld by Platt.[5] He states that the condition is often associated with pyrexia, enlarged tonsils, or chronic ear disease, and often with a certain amount of ill-health. He holds the view that the disease is hæmatogenous in origin like other forms of osteomyelitis. He considers that the area of the femoral neck supplied by the upper leash of metaphysial vessels is that first affected, since that region is more vascular than the synovial membrane at the age when pseudocoxalgia is found. He explains the absence of the disease in older patients by the relative increase in the vascularity of the synovial membrane with age, which makes the joint proper more likely to suffer than the metaphysial region, and therefore the changes in the head we associate with pseudocoxalgia do not occur.

As we have already noticed, exploration of the joint has rather upheld the infective theory. Anyone who has seen cases of pseudocoxalgia in the active stage with marked pain, muscle-spasm, and limitation of movements, cannot help being impressed by the clinical resemblance to infective arthritis. It is well known that coxa plana occurs in lesions of the metaphysis known to be infections, e.g., tuberculosis.

Against the idea that the condition is due to an infection are these facts: abscesses never develop, there is no leucocytosis, the neighbouring structures are never affected, the radiographic appearances precede the clinical signs (contrast infected joints), and except in rare instances the femoral epiphysis is the only one affected.

Developmental Theories.—About the end of the second month of fœtal life the human hip-joint resembles that of adult reptiles. There is an imperfectly formed and shallow acetabulum and a small malformed head. The limb is then at right angles to the axis of the body and rotated outwards, the extensor surface of the knee being directed upwards and the great toe in front. If rotation occurred at this time, to enable the erect position to be assumed, dislocation would be likely to take place.

Calot[13] thinks that in pseudocoxalgia there is a condition of arrested

development of the hip-joint such as that just described, with a tendency to congenital dislocation of the hip, and that the result of this is that the head fits imperfectly into the acetabulum, with resulting osteochondritis from the mechanical disability.

Mürk Jansen[14] has propounded a somewhat elaborate theory to account for the condition. He says that the curve of the acetabulum is flatter than the curve of the femoral head in patients who develop pseudocoxalgia, and that this is caused either: (1) By the bony floor of the acetabulum being too thick; or (2) By the ischium being bent inwards towards the mid-line, a condition of 'ischium varum'; and he states that these abnormalities are produced by a small amniotic sac which either tends to flex and adduct the hip-joint and lever the head out of the acetabulum and so lessen the normal pressure of the head against the acetabular floor; or else, by being tightly drawn over the buttock, bends the ischium inwards.

If the difference of curvature produced in either of these two ways be granted, Jansen says that the mechanical conditions which follow will naturally result in changes in the head of the femur.

The following objections have been raised to this theory:—

1. That ischium varum is seen if the radiograph be not taken exactly symmetrically, and that it occurs in many conditions without pseudocoxalgia following.

2. That in many cases pseudocoxalgia occurs without any discrepancy in the curvature of the head and acetabulum.

3. That if the condition were due to pressure on only a small part of the head, early cases treated by traction and fixation should be arrested.

In reply to this last objection, Jansen says that so long as any discrepancy of joint surfaces exists, abnormal stimuli will be acting, and that the measures of treatment adopted are insufficient to prevent this.

4. The theory has also been criticized on the grounds that pseudocoxalgia often occurs some years after the child has begun to walk. Jansen, however, asks us to consider other deformities, such as genu valgum, which also take time to manifest themselves.

The Traumatic Theory.—The epiphysis of the upper end of the femur is almost entirely dependent for its blood-supply on the small vessels entering its circumference where the head and neck join. At birth the upper epiphysis of the femur includes the great trochanter; later the neck of the femur develops as an upward extension of the shaft which eventually divides the epiphysis into two: (*a*) the head, (*b*) the great trochanter. This division is complete by the third year, which is the earliest age at which pseudocoxalgia has been noticed, and is the age at which there is for the first time great restriction in the blood-supply to the head. It is thought that a slight injury may result in obliteration of some of the vessels supplying the femoral epiphysis, with atrophy of bone-cells and decalcification. The cartilage, which gets its nutrition from the synovial fluid of the joint, remains unaffected; but with the disappearance of its bony nucleus the cartilage collapses; eventually, when the vascular supply is restored, new bone is laid down but the epiphysis is misshapen and deformed.

Recently Bentzon,[15] by injecting alcohol round the arteries of the upper

epiphysis of the femur in rabbits and so paralysing the vasoconstrictor nerves, has produced coxa plana, which he considers is caused by the active hyperæmia so induced. He thinks that the arteries supplying the caput femoris are especially exposed to mild lesions in young people, where injury may bend the cartilaginous parts of the epiphysis.

Legg[16] tunnelled into the neck of a femur for a definite acute septic condition; five years later flattening of the epiphysis occurred, due he considers to interference with the blood-supply during the operation.

TREATMENT.

There is a tendency towards spontaneous recovery in this condition, and patients never ultimately show complete incapacity; on the other hand, the end-results vary considerably so far as the function of the limb and comfort of the patient are concerned.

In the first case it is only fair to state that quite a number of observers consider that treatment has no effect on the condition. Legg[2] sums up the views of these surgeons when he says: "While a process suggesting weakness of bone structure is going on it is theoretically sound to allow no weight-bearing; but in practice, relief from weight-bearing in no way affects the end-result." The majority of surgeons, however, consider that if treatment be begun before the cartilaginous envelope of the epiphysis has been deformed, the subsequent malformation will be much less marked, although the fragmentation and cycle of bony changes will not be completely averted. An examination of my own cases bears out this view.

The treatment most commonly recommended is fixation in an abducted position, and measures to prevent the weight of the body being borne by the diseased limb. Some surgeons[9] think that movement does no harm if the latter point be attended to. The duration of fixation necessary should be controlled by repeated X-ray examination to determine the degree of recalcification reached by the head of the bone, and may be any length of time between six months and three years.

In cases with a large amount of muscular spasm and pain, one is forced to treat the condition for the sake of the patients' comfort even if one has not much faith in its effect on the end-results. In these acute cases fixation is insufficient to relieve the pain, and some form of extension apparatus is necessary to overcome the muscular spasm.

Kidner[10] and others[11] have operated on patients with this disease, on the assumption that it is a mild form of osteomyelitis. Kidner scraped out the débris from an area of rarefaction in the neck of the femur of a boy of 5. He observed by X rays the subsequent healing, and said that the operation hastened this and diminished the subsequent deformity. The great majority of surgeons, however, consider an operation of this sort to be unjustifiable.

Many years after the disease is apparently cured, pain and stiffness may develop. These cases may or may not show X-ray evidence of osteo-arthritis; in the latter instance operation to remodel the head may be desirable.

DETAILED REVIEW OF 16 CASES OUT OF 25 TREATED.

CASE 1.—*Male. Age at onset, 6 years 7 months.*

FIG. 62.

FIG. 63.

FIG. 64.

March 17, 1924 (*Fig.* 62).—Only movement possible was slight flexion. No pain. Metaphysial rarefaction is well shown.

July 28, 1924 (*Fig.* 63).—Rotation, inversion, and abduction limited, other movements perfect. Treatment: weight-extension since admission.

Feb. 18, 1925.—Six months after discharge, without treatment, movements were perfect.

Jan. 26, 1926 (*Fig.* 64).—Seventeen months after discharge. No symptoms.

CASE 2.—*Female. Age at onset, 3 years 4 months.*

FIG. 65.

FIG. 66.

FIG. 67.

FIG. 68.

FIG. 69.

FIG. 70.

May 27, 1922 (*Fig.* 65).—Limp began 10 months before and has been intermittent. Now some pain and all movements limited, can walk with limp. During the 10 months since onset of first symptoms the head of the femur has practically disappeared.

Dec. 11, 1923 (*Fig.* 66).—Patient has been treated in bed for 7 months in a short hip-spica plaster. All movements except internal rotation and abduction are perfect; no pain. Patient was discharged from hospital with a light celluloid splint, crutches, and patten. Note metaphysial rarefaction.

May 5, 1924 (*Fig.* 67).—Movements nearly perfect. Splint removed, crutches and patten continued for further 6 months.

March 13, 1925 (*Fig.* 68).
Feb. 6, 1926 (*Fig.* 69).
Sept. 8, 1927 (*Fig.* 70).
} Further stages of repair. *Fig.* 69 shows condition of head 2 years 10 months after treatment ceased.

CASE 3.—*Male. Age at onset, 7 years 6 months.*

FIG. 71.

FIG. 72.

FIG. 73.

Sept. 3, 1924 (*Fig.* 71).—Disease followed directly after measles 1 month ago. Attempt at movement in any direction causes considerable pain. Treated for 6 months with weight-extension and then for 3 months in a plaster splint. Note metaphysial rarefaction.

July 8, 1925.—Movements nearly perfect, no pain. No treatment except bed.

Dec. 10, 1925 (*Fig.* 72).—Discharged from hospital with no retentive apparatus. Movements perfect.

Aug. 4, 1927 (*Fig.* 73).—One year and 8 months after discharge from hospital. Movements perfect. Slight limp when tired.

CASE 4.—*Male. Age at onset, 8 years 9 months.*

FIG. 74.

Dec. 30, 1926 (*Fig.* 74).—At first examination 6 months after onset of limp, had been treated as tuberculosis of hip. *Left hip*: very acute disease, marked pain, no movements possible. *Right hip*: no symptoms. Note metaphysial changes.

July 17, 1927 (*Fig.* 75).—Left hip: marked limitation of internal rotation and abduction. Marked limp, no pain.

FIG. 75.

CASE 5.—*Male. Age,* 10 *years.*

FIG. 76.

FIG. 77.

April 10, 1923 (*Fig.* 76).—Duration: right, unknown; left, 2 years. Right, no symptoms; left, slight muscle-spasm and pain. Treatment: fixation 9 months, and then home without apparatus.

Nov. 7, 1926 (*Fig.* 77).—Perfect movements both hips.

FIG. 78.

CASE 6.—*Male. Age, 18 years.*

First seen March, 1916; had had intermittent pain and limp for 18 months.

Fig. 78.—Shows condition 11½ years after first examination. Was treated nearly 2 years by fixation, and for 6 months with no weight-bearing. Some limitation of abduction and slight limp, but patient can walk twenty miles.

CASE 7.—*Male. Age at onset, 4 years 4 months.*

FIG. 79.

FIG. 80.

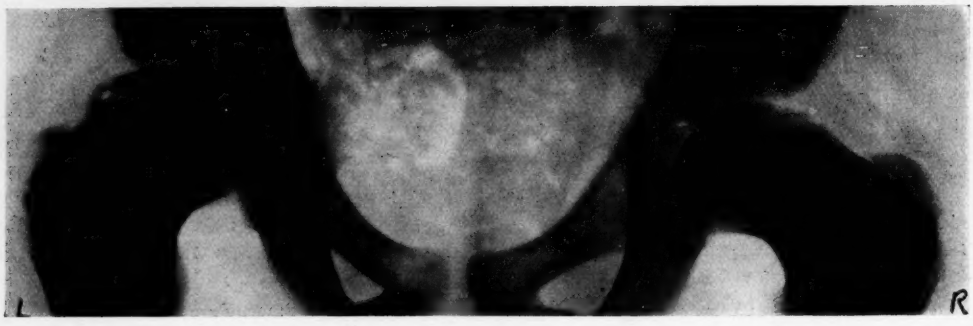

FIG. 81.

Feb. 8, 1923 (*Fig.* 79).—Taken at time of patient's first appearance. He came because of pain in the *right* hip, with considerable muscle-spasm. The *left* hip disease was not associated with symptoms, although at an earlier and more active stage than the right radiographically. Note metaphysial rarefaction (left).

March, 1924 (*Fig.* 80).—Patient has had 1 year's fixation of both hips; now no symptoms. Epiphyses of both femora are beginning to re-form. Now no treatment.

Aug., 1927 (*Fig.* 81).—Three years and 5 months after all treatment has been discontinued, and 4 years and 6 months after first examination. Movements are only very slightly diminished; the left hip in the direction of internal rotation, and the right in abduction. Very slight limp and no pain.

CASE 8.—*Female. Age at onset, 5 years.*

FIG. 82.

FIG. 83.

FIG. 84.

March 4, 1922 (*Fig.* 82).—First examination: slight occasional pain in left hip when tired, for nearly 1 year. Treatment: fixation 6 months, and then bed without apparatus 10 months. Note metaphysial rarefaction (left). Destruction of epiphysis has progressed.

July 5, 1923 (*Fig.* 83).—No symptoms in either hip. Sent home without apparatus. No limp and no pain.

June 8, 1925.—After 23 months at home, re-examined with history of slight pain in *right* hip for a week, with marked limp and limitation of movement. Treatment: 1 month fixation, and then discharged with slight limp and without apparatus.

Aug. 5, 1927 (*Fig.* 84).—Two years and 2 months after discharge. Left hip movements perfect. Right hip, very slight limitation of abduction and internal rotation.

CASE 9.—*Male. Age at onset, 9 years 4 months.*

FIG. 85.

FIG. 86.

FIG. 87.

Jan., 1925 (*Fig.* 85).—History of 4 months' slight pain. Movements good. Treated for three months with extension, then no treatment.

Jan. 1, 1926 (*Fig.* 86).

Aug., 1927 (*Fig.* 87).—Two and a half years after first examination, and two years and four months after discharge. Practically no limp.

CASE 10.—*Male. Age at onset, 5 years 3 months.*

FIG. 88.

FIG. 89.

March 6, 1922 (*Fig.* 88).—History two months; only symptom slight limp. Note metaphysial rarefaction. Treatment: extension, four months; plaster, 12 months; walking with splint, crutches, and patten, 17 months.

Aug., 1927 (*Fig.* 89).—Three years and a half after treatment stopped. Movements perfect, no limp.

CASE 11.—*Female. Age at onset, 7 years 9 months.*

FIG. 90.

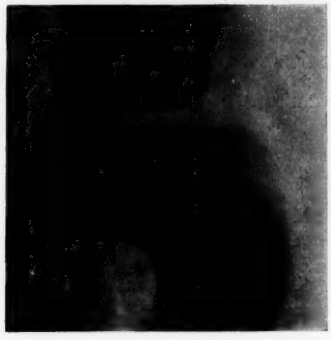

FIG. 91.

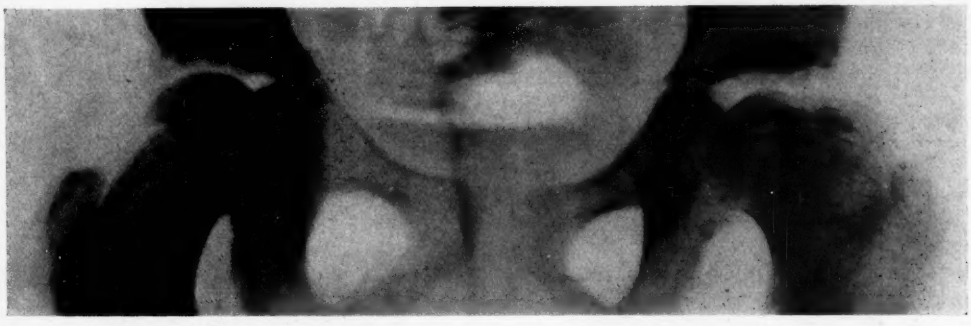

FIG. 92.

Jan. 2, 1924 (*Fig.* 90).—Limping one week, pain in left hip and knee, 1 in. wasting of thigh. Movements all good. No history of trauma. Admitted as tuberculosis and fixed for 9 months, then diagnosed as pseudocoxalgia ; discharged with slight limitation of abduction only, and allowed to use limb.

April 16, 1925 (*Fig.* 91).—Six months after discharge, re-admitted with pain, left hip slightly flexed and adducted, movements all limited. Fixation for 8 months, and then no weight for a further 2 months. Note metaphysial rarefaction.

Aug. 10, 1927 (*Fig.* 92).—One year and a half after discharge. Slight abduction, slight limp when tired ; otherwise no symptoms.

CASE 12.—*Male. Age at onset: left hip, 5 years 5 months; right hip, not known.*

FIG. 93.

FIG. 94.

Feb., 1923 (*Fig.* 93).—For 5 months slight pain in *left* hip, no symptoms in *right*. On examination, marked pain and muscle spasm, with no movements. Fixed for eight months, then no treatment. Metaphysial rarefaction well shown in both.

Aug., 1927 (*Fig.* 94).—No treatment for 3 years 10 months. Movements perfect; very slight limp only after a long walk.

CASE 13.—*Male. Age at onset, 5 years.*

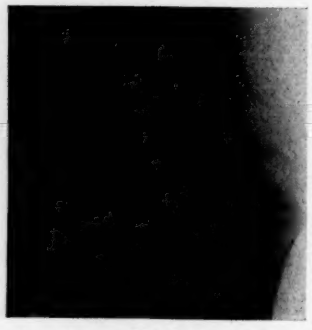

FIG. 95.

FIG. 96.

Jan. 4, 1925 (*Fig.* 95).—Slight pain and limp 1 year; now definite pain and spasm. Treatment: 3 months with fixation, then none.

Aug. 3, 1927 (*Fig.* 96).—After 2 years and 4 months without treatment. No signs or symptoms.

CASE 14.—*Male. Age at onset, 4 years 8 months.*

FIG. 97.

FIG. 98.

July, 1925 (*Fig.* 97).—Six months' history. Very active disease; pain and spasm marked. Treated by fixation 4 months; all symptoms disappeared. Discharged without apparatus.

Aug., 1927 (*Fig.* 98).—After 20 months without treatment; abduction markedly limited, with a certain amount of resulting limp; otherwise no symptoms.

CASE 15.—*Female. Age at onset, 10 years.*

FIG. 99.

FIG. 100.

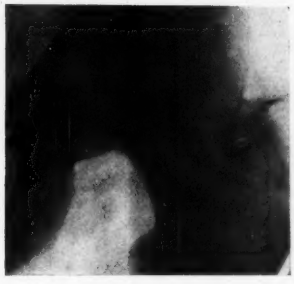

FIG. 101.

Aug., 1924 (*Fig.* 99).—History of slight pain occasionally, 1 year. Some flexion possible, but no other movements. Hip adducted and slightly flexed. Pain considerable.

Feb., 1925.—Disease progressing slowly in spite of treatment by fixation; no pain and limitation of abduction, and internal rotation only.

June, 1925 (*Fig.* 100).—A further stage of the disease. Treatment: fixation from admission for 2 years and 2 months, and then no treatment.

June, 1927 (*Fig.* 101).—Eight months after discharge. No symptoms; slight limitation of abduction.

FIG. 102.

CASE 16.

Oct., 1927 (*Fig.* 102).—End-result 6 years after onset. Limitation of internal rotation and abduction. Limp. Was treated for 9 months during active stage.

SUMMARY OF 25 CASES, WITH SPECIAL REFERENCE TO THE END-RESULTS.

CASE	FIGURE NUMBERS	DEGREE OF ACTIVITY ON FIRST EXAMINATION	TREATMENT (in months)		END-RESULTS (Movements limited and limp)
			Fixed	Not fixed, but no weight-bearing	
1	62–64	Marked muscle-spasm. No pain	5	–	Perfect movements
2	65–70	Some spasm and pain ..	12	6	Internal rotation (slight)
3	71–73	Marked pain and spasm ..	9	4	Perfect movements, slight limp when tired
4	74, 75	*Left:* Marked pain and spasm	4	2	Internal rotation and abduction (marked)
		Right: No symptoms ..	6	–	Considerable limp
5	76, 77	*Left:* Very slight pain and spasm	9	–	Perfect movements
		Right: No symptoms ..	–	–	
6	78	Slight pain and spasm ..	20	6	Abduction, slight limp
7	79–81	*Left:* Pain and spasm ..	12	2	Internal rotation (slight)
		Right: No symptoms ..	–	14	Abduction (slight)
8	82–84	*Left:* Slight pain and spasm	3	3	Perfect movements, no limp
		Right: Pain and spasm very marked	1	6	Perfect movements, no limp
9	85–87	Some pain	3	–	Perfect movements, no limp
10	88, 89	No symptoms	16	7	Perfect movements, no limp
11	90–92	Pain and spasm (moderate)..	12	6	Abduction, slight limp
12	93, 94	*Left:* Pain and very marked spasm	8	–	Very slight limp when tired
		Right: No symptoms ..	1	–	Perfect movements
13	95, 96	Pain and spasm moderate ..	3	2	Perfect movements, no limp
14	97, 98	Pain and spasm marked ..	4	2	Abduction very limited, limp
15	99–101	Pain and marked spasm ..	28	–	Perfect movements, no limp
16	102	Very slight pain and spasm ..	3	6	Abduction, internal rotation, slight limp
17	—	No symptoms	2	3	Perfect movements, no limp
18	—	Marked pain and spasm ..	2	–	Abduction, internal rotation, slight limp
19	—	Some spasm, no pain ..	6	4	Perfect movements, no limp
20	—	Pain and spasm very slight	8	4	Internal rotation (slight)
21	—	Some pain and spasm ..	10	2	Abduction and marked limp
22	—	Some spasm, no pain ..	8	–	Internal rotation, abduction, slight limp
23	—	Pain and spasm	30	–	Perfect movements, no limp
24	—	Pain and marked spasm ..	6	4	Internal rotation, and limp
25	—	Slight spasm, no pain ..	9	3	Perfect movements, no limp

CONCLUSIONS.

1. The majority of patients with pseudocoxalgia have some degree of pain and muscular spasm some time during the early stages of the disease.

2. The initial degree of activity of the disease does not necessarily bear any relation to the ultimate functional result.

3. The ultimate function of the affected limb is very good in the majority of cases. In patients in whom movement is limited the limitation is in the direction of abduction and internal rotation.

4. The length of treatment required to obtain a good result varies greatly, and does not depend on the initial amount of activity of the disease.

I desire to express my thanks to Dr. Veronica Dawkins for the preparation of the reproductions of the skiagrams in this paper.

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STAPHYLOCOCCAL SUPPURATIVE NEPHRITIS (CARBUNCLE OF THE KIDNEY).

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The term 'carbuncle of the kidney' has been in use for many years to describe an uncommon, but yet definite, type of suppuration of the kidney. The chief characters of the lesion denoted by this term are briefly as follows: There is an acute or subacute hæmatogenous infection of the parenchyma of one kidney by the *Sta. aureus*, the onset of which is frequently preceded by some trauma to the organ. The source of origin of the organism is most often a skin lesion such as a whitlow, boil, or carbuncle. The appearance of the kidney varies according to the stage at which the disease is encountered by the surgeon or pathologist; the most constant finding is that of multiple areas of necrosis or suppuration in the substance of the kidney, confined to one area of it. Fusion of the suppurative foci gives rise to a honeycomb-like necrotic cavity, or a circumscribed encapsulated abscess, and a localized enlargement of the affected area of the kidney. A perinephric abscess may result from the outward extension of the suppurative process, but eruption into the renal pelvis is unusual.

Review of the recorded cases of this condition indicates that it deserves to rank as a clean-cut pathological and clinical entity, and that its diagnosis may be arrived at with considerable accuracy. In one of the cases here recorded, recognition of the disease was facilitated by the pyelographic appearance presented by the kidney. The extent of the suppurative process and its relation to the structures of the kidney are demonstrated clearly by the accompanying coloured illustrations.

CASE RECORDS.

Case 1.—A young man, age 23, was admitted to hospital on account of pain in the right loin.

History.—A month before admission, while on a motor cycle, the patient had an attack of gnawing pain in the right loin which lasted for a few minutes. Two days later a similar attack was experienced, and he began to feel 'out of sorts'. From that time onwards he had repeated attacks of pain, which were at first slight, became more severe and continuous, and came on several times during the day, so that he was compelled to remain in bed. The patient was very feverish and uncomfortable, and on two or three occasions had rigors which were attended by nausea and vomiting. He got some ease by drawing the right thigh up. Shortly before admission to hospital, micturition had become more frequent, especially at night, but no alteration in the appearance of the urine had been observed. The patient

had lost his appetite, felt languid, and had become very thin; he sweated a great deal and felt his mouth always dry. He was very constipated. Two months before the onset of his illness a tooth had been extracted on account of an abscess at its root.

On Admission.—The man was very emaciated and looked ill. His face was pale and his lips dry and cracked. For comfort he lay with the right hip flexed. Temperature 99°, pulse-rate 92. Examination of the abdomen elicited the presence of a tender ill-defined swelling in the right loin which moved with respiration.

The patient was under observation for two weeks before an operation was performed. During this period the temperature fluctuated from 99° to 102°; the pulse-rate remained about 90. The leucocyte-count was 12,000 per c.mm. Microscopic examination of the urine was carried out many times, but only on two occasions were pus-cells detected, and then only a few.

Fig. 103.—*Case* 1. Pyelogram of right kidney. Note the great reduction in volume of the renal pelvis and major calices. The minor calices are narrowed and deformed, and have assumed a 'frond-like' appearance.

Urological and Radiographic Examination. — On cystoscopic examination the bladder walls were seen to be healthy, and both ureters to secrete freely. Samples of urine collected from the right kidney, which was under suspicion, were sterile and contained no pus-cells. Each renal pelvis was injected with 14 c.c. sodium iodide, very slight discomfort being induced. The left pyelogram was normal, but the right showed a very striking alteration in form; the appearance is well demonstrated in *Fig.* 103, which shows that the lower major calix is very much narrowed, and that its minor calices have assumed a frond-like appearance. The upper calix showed a similar but less marked change.

Operation.—A perinephric abscess was found at the lateral border of the right kidney. About a teacupful of pus (which subsequent culture showed had been produced by *Sta. aureus*) was evacuated. The abscess communicated with the interior of the kidney by a small aperture on its lateral border; therefore the kidney was removed. Recovery was slow but satisfactory.

Appearance of the Kidney.—The middle and lower part of the kidney were uniformly enlarged, and the capsule was a little thickened and adherent

over the swollen area. Below the centre of the kidney and at its lateral border there was a small ragged-edged opening from which purulent and necrotic material could be expressed. On section, the lower half of the cortex and medulla of the kidney were found to be the site of multiple areas of suppuration, which in places were separate and sharply defined, but in other parts had become confluent and produced a spongy and irregular zone of necrosis. The lumen of the renal pelvis and calices was greatly narrowed (and in places constricted) by the adjacent inflammatory process. Penetra-

FIG. 104.—*Case* 1. Cut surface of kidney. The left figure shows an irregular necrotic mass in the kidney substance; calices are seen traversing the carbuncular area. The figure on the right shows multiple foci of necrosis, which, in places, are discrete and almost encapsulated; in other parts, suppurative areas have coalesced and broken down. Small concentric foci of necrosis are seen in the upper part of the kidney.

tion of the suppurative process into the calices could not be detected in large sections which were made of the whole kidney at different depths. Microscopic examination showed that the inflammatory foci were demarcated from the normal kidney tissue by a ring of newly-formed connective tissue. ***Fig.* 104** demonstrates the resemblance of this type of renal suppuration to that found in a carbuncle of the skin.

Case 2.—A well-developed youth, a miner, age 17, was admitted to hospital, under the care of the late Professor Alexis Thomson, for pain in the left loin.

History.—For three weeks before he was sent into hospital he had felt weak and listless; the least exertion caused him to feel tired; and his appetite had become very poor. A week prior to coming to hospital, while at work, he was seized with acute pain below the ribs on the left side. The pain, which remained in the same position, got gradually worse, and he had to give up work and go to bed. Relief was obtained by a hypodermic injection of morphine. On the following day the pain was less severe, but he had a constant gnawing sensation in the left loin. Pressure or movement excited an attack of acute pain. He was most comfortable when he lay on his right side. The patient required to void his urine about ten times in twenty-four hours; his bowels were stubbornly constipated.

On Admission.—The patient was of good physique, but looked pale and ill. The tongue bore a thick brown fur, and the mouth and lips were dry. No septic lesion of the skin, mouth, or pharynx was discovered. Examination of the abdomen revealed very marked tenderness over the left kidney region and in the upper part of the left iliac fossa; tenderness on pressure was experienced also in the kidney angle behind. The left kidney could not be palpated. The temperature was 102·5°, and the pulse-rate 90.

The patient was under observation for nearly a month before an operation was performed; during this time the temperature varied from 99° to 102°, and the leucocyte-count ascended from 11,000 to 14,000 per c.mm. The urine contained neither pus, blood, nor organisms until a few days before operation, when a few leucocytes and red blood-corpuscles were found under the microscope. The discovery of these elements in the urine hastened the decision to explore the left kidney.

Fig. 105.—*Case* 2. Appearance of bisected kidney. The centre and upper part of the kidney contains a spherical mass of necrotic tissue. Note the encapsulation by newly-formed connective tissue, and the relation of the swelling to the upper calix of the kidney, which is narrowed and elongated.

Operation.—The left kidney was explored by the usual oblique lumbar incision. The organ was found to be a little enlarged, and an ovoid swelling in the kidney substance was detected at its upper pole. The kidney was removed, and a rapid recovery ensued.

Appearance of the Kidney on Section.—An abscess, which was circumscribed by a thick ring of newly-formed fibrous tissue, occupied the upper and middle part of the kidney substance. The abscess extended outwards to within a quarter of an inch of the surface of the kidney. In the region of the hilum of the kidney the abscess was in contact with the outer wall of the upper major calix, which was narrowed and elongated (*Fig.* 105).

Microscopic sections taken from the abscess wall showed a thick layer of newly-formed connective tissue that had enveloped the broken-down inflammatory products. Sections stained by Gram's method demonstrated abundant staphylococci in the interior of the abscess.

Case 3.—A married woman, age 48, came into hospital, under the care of Sir Harold Stiles, on account of pain in the right loin and severe debility.

History.—The patient had been in robust health until two months before her admission. Her complaint began fairly suddenly with a feeling of great tiredness which made her unfit for her household duties. She had severe frontal headaches, a 'chilly' feeling in the evenings, and great difficulty in getting her bowels to act. After a month she began to experience discomfort and constant aching in the right side below the costal margin in front. Attacks of severe stabbing pain, which did not radiate in any particular direction, began to be experienced; in the intervals, gnawing lumbar pain persisted. Pain was increased by any sudden movement or by deep inspiration, as, for example, a dry cough which supervened a week before she came to hospital. During this month she had frequency of micturition, especially at night. No change in the appearance of the urine had been noticed by herself or by her doctor.

Fig. 106.—*Case* 3. Cut surface of left kidney. The upper part of the kidney is replaced by a cavernous necrotic mass. The remainder of the kidney is normal in appearance.

On Admission.—The patient was emaciated and looked ill; her complexion was muddy and her skin dry. The tongue bore a thick brown fur. Attempts to palpate the right kidney produced excessive tenderness both in front and behind. Pressure over the lower part of her thorax in front caused pain. The kidney could not be felt. On auscultation, the breath-sounds were much fainter at the base of the right lung than the left. The temperature was 99°, and the pulse-rate 100.

The patient was kept under observation for ten days, during which period the pain was at times severe and she became more ill. The temperature was intermittent, with an evening rise to about 101°, and a morning fall to subnormal. Microscopic examination of the urine from time to time showed numerous pus-cells, and as a result of culture a growth of *Sta. aureus* was obtained.

Operation.—Although the pyuria was insufficient to cause naked-eye change in the urine, the case was considered to be one of pyonephrosis; operation was accordingly carried out, and the kidney removed. It was

enlarged and pyriform; the enlargement was confined to the upper pole, which felt soft and boggy, and a fluctuating swelling was palpable at its anterior surface. The capsule of the kidney was greatly thickened, and adhesions to neighbouring viscera made its removal a little difficult.

The patient made a satisfactory recovery except for the development of an apparently unrelated ischiorectal abscess which delayed convalescence for some time.

Appearance of the Kidney on Section.—The upper pole was replaced by multilocular abscess cavities which contained thick pus and necrotic material. The walls of the abscesses were lined by shreds of yellow sloughs. Areas of less advanced necrosis surrounded the main abscess cavities (*Fig.* 106). Intact but much narrowed and thickened calices traversed the diseased part of the kidney; the insertion of a fine probe along the lumen of the calices showed that their walls had resisted the invasion of the suppurative process.

The bisected kidney bore a striking resemblance to renal tuberculosis, and the true nature of the disease was only recognized after a pure culture of *Sta. aureus* had been grown from the contents of the abscess cavities. The staphylococci were stained *in situ* in sections taken from the diseased part of the kidney.

ETIOLOGY.

Some twenty-seven cases of carbuncle of the kidney are reported in the literature. The condition has received scanty recognition by British writers. The disease attacks men oftener than women; more frequent exposure to trauma is held responsible for its greater incidence in the male sex. Trauma, usually of the nature of a strain, is supposed to lower the resistance of the kidney and render it more vulnerable to organisms that, in a normal organ, would be eliminated without producing ill-effects (Thompson[1]). This belief is supported by the experimental work of Brewer;[2] he found that the injection of bacteria into the circulation of 16 animals that had received trauma to one kidney resulted in the development of suppurative nephritis in the damaged kidney of 11 of the animals, and that the inflammatory changes began in the interstitial tissue of the kidney.

In carbuncle of the kidney the commonest portal of entry into the circulation for the *Sta. aureus* is a *skin* carbuncle, boils, or smaller multiple septic skin foci. Many other foci, such as a whitlow, a patch of eczema, and acute osteomyelitis, have been described. In *Case* 1 of this report a 'root abscess' was considered to be the source of infection. Horn[3] relates a case in which the disease followed an operation for removal of the prostate. The primary focus may be overlooked or not discovered, and it is of interest to note that symptoms of renal involvement may not appear until several weeks after the peripheral lesions have become quiescent, or have actually healed.

The earliest age at which the disease has been known to occur is 10 years (Barth,[4] Colmers[5]), and the oldest 55 years (Reschle[6]). Only in one recorded case was the infection bilateral (Kretschmer[7]); in that patient, signs of involvement of the second kidney appeared ten days after nephrectomy had been performed on the other side.

CLINICAL FEATURES.

Symptoms come on gradually with malaise, headache, and loss of appetite; the temperature is raised, and the patient may have repeated rigors, and he may vomit. Pain may be present from the outset, but often does not appear for a considerable time after the other symptoms (*Cases* 2 and 3). The pain is of a constant gnawing character, and is confined to the loin, the lower part of the chest, and the side of the upper abdomen; sharp exacerbations, excited by sudden movements or deep inspiration, occur from time to time. For comfort, the thigh on the affected side may be kept in a state of flexion. Urinary symptoms are generally absent; nocturnal frequency, and the passage of dark concentrated urine, may be noticed by the patient. As the disease progresses, the patient becomes extremely ill, sweats profusely, and becomes very emaciated. An irritating dry cough may develop. The bowels are very constipated.

The findings on physical examination will vary according to the duration of the illness. The abdominal muscles and the lower part of the thorax on the affected side show diminished respiratory movement, and a localized fullness or actual swelling may be seen in the loin. On palpation, defensive boarding of the lumbar and abdominal muscles is felt, and a very tender ill-defined mass may be detected in the lumbar region. The leucocyte-count ranges from 10,000 up to 25,000 per c.mm. Microscopic examination of the urine is most often negative, and catheter specimens are usually sterile; a few leucocytes and red blood-cells may be found from time to time, but are never abundant.

DIAGNOSIS.

A valuable aid in the diagnosis is the discovery that the patient has recently suffered from a septic skin lesion.

Pain in the lower part of the chest and fixation of the diaphragm on that side may lead to a suspicion of pleurisy or pneumonia; careful physical examination should be sufficient to exclude an intrathoracic lesion. An accurate history and careful abdominal examination should prevent confusion with retrocæcal appendicitis. In acute metastatic abscesses, in which the whole kidney is studded with small areas of suppuration, the urine usually contains a large quantity of pus and bacteria.

Pyelography should prove useful in determining whether a perinephric abscess has arisen from a renal carbuncle; discovery of a filling-defect in one or more calices would suggest an intrarenal origin of the abscess, and, in addition, would serve to decide the correct line of surgical treatment.

TREATMENT.

Nephrectomy is the most certain way of eradicating the disease, and when the operation is performed early the prognosis is good and recovery prompt. When the carbuncle is small, and restricted to one pole of the kidney, resection of the affected area has been recommended; cases will be few in which this method of treatment is practicable. Drainage of an actual abscess seen at operation has sometimes been successful.

In some of the reported cases a perinephric abscess had been drained and the renal origin of the infection overlooked or not suspected. In those cases, benefit was only temporary and fever soon returned; a secondary nephrectomy gave relief, and disclosed the origin of the perinephric abscess.

I should like to thank Mr. J. M. Graham for permission to publish the details of *Case* 1. I am obliged to Professor D. P. D. Wilkie for permission to make use of the specimen of the kidney of *Case* 2, and to Professor John Fraser for a similar privilege in *Case* 3. The urological investigation of *Case* 1 was carried out in the Electrical Theatre of the Royal Infirmary, Edinburgh. The expense of production of the coloured illustrations was defrayed by a grant from the Medical Research Council, to whom I render my thanks.

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OBSERVATIONS ON CALCIFICATION OF THE GALL-BLADDER: WITH THE PRESENTATION OF A CASE.

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Calcification of the gall-bladder is a very rare condition, if we may judge from a search in the literature or in the museums. It seems worth while therefore to describe a new case of this condition and to attempt a classification of those specimens found in the Museums of the Royal College of Surgeons in London and Edinburgh.

Early in the perusal of the literature bearing on this subject it becomes evident that in fully 90 per cent of cases calcification of the gall-bladder is accompanied by gall-stones, and as the case to be recorded suitably exemplifies this, it will now be described.

CASE REPORT.

A. B. was admitted to the Bristol General Hospital under the care of Mr. Moore. The patient is the mother of eleven children. Prior to the past eleven months she had been in perfect health, but since then her appetite has become poor. She has been subject to attacks of nausea without vomiting. In her right subcostal region she has had an almost continuous gnawing pain, also felt in her back around the medial angle of the scapula. This aching pain was increased by the ingestion of any food, fluid or solid. She has had no attack of jaundice.

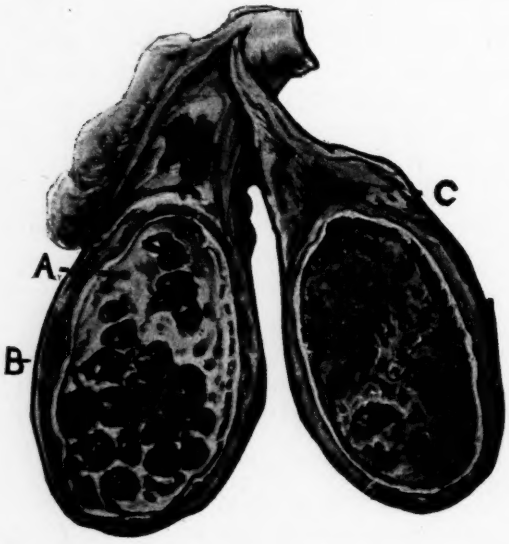

Fig. 107.—Calcareous gall-bladder. A, Calcium salts; B, Calcareous area; C, Cystic duct opened.

On Examination.—A well-nourished woman, looking in perfect health. Abdominal inspection reveals tenderness over the gall-bladder. Nothing is palpable.

Operation.—The gall-bladder was approached through a right paramedian incision and found to be hard, and fixed by some periglandular adhesions. Its serous coat was thickened. Nothing abnormal

could be felt in the hepatic or common bile-ducts. The remaining structures in the abdomen were healthy. Cholecystectomy was performed. The patient made an uninterrupted recovery, and left hospital twenty-one days after her operation.

PATHOLOGICAL EXAMINATION (*Fig.* 107).—In shape the gall-bladder is that of a walnut, measuring from fundus to the entrance to the cystic duct 3·6 cm., and the greatest transverse diameter 2·6 cm. The serous coat is of a dull-grey colour, thickened and fibrotic. It can be stripped cleanly from the subjacent gall-bladder wall. Macroscopically the middle or muscular layer has the appearance of, and cuts like, bone. In thickness it averages 0·2 cm. The mucosa has entirely disappeared, and in its place is a layer of lime salts which completely encircles the wall of the bladder, crossing over and shutting off the entrance to the cystic duct inferiorly, but not extending into it. The gall-bladder is completely filled with laminated calculi, varying in size, the largest measuring 1·5 cm. across. These are rigidly cemented

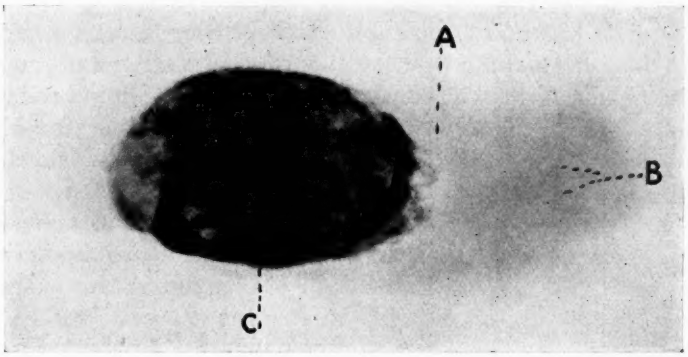

FIG. 108.—Radiogram of the gall-bladder taken after removal. A, Collum; B, Cystic duct C, Corpus.

together by the interposition of lime salts. The cystic duct shows no sign of calcification, nor does its lumen contain any calculi.

X-RAY EXAMINATION (*Fig.* 108).—A radiogram of the gall-bladder taken after removal shows the marked degree to which calcification has occurred, and the density of the calcareous matter deposited in the muscular layer of the wall. Again, it demonstrates the abrupt ending to the calcification immediately at the entrance to the cystic duct. X-ray photographs were also taken with a view to finding areas of sclerosis in other parts of the patient's body, but none were discovered.

MICROSCOPIC EXAMINATION.—A decalcified section from the gall-bladder wall was stained by hæmalum and eosin, iron hæmatoxylin and Van Gieson, Mallory's aniline blue and fuchsin. The mucosa is entirely absent. The muscular stratum has been wholly replaced by an acellular homogeneous substance—the decalcified layer. There is no evidence to suggest that this is of the nature of true bone, or even progressing towards bone formation. The inner and outer margins of this layer are well defined, and externally it is outlined by a thick coat of fibrous tissue.

OBSERVATIONS.

In this case the calcified gall-bladder is filled with calculi, these being cemented closely together with hard calcium salts, the whole surrounded by a thickened and fibrosed serous lining. The calcification of the wall is confined entirely to the parts surrounding the calculi, and does not extend beyond them in the direction of the cystic duct. This relationship is extremely significant, and is well substantiated by the case presented by Talbot. There the gall-bladder and the first part of the cystic duct both contained calculi and both were calcified, but the last inch of the cystic duct contained no calculi and was normal in appearance and size. It would seem therefore that a relationship exists between the calculi and the calcareous change in the adjacent wall. Realizing that the functional relationship is that of a contractile sac to an unyielding content, the sequence of pathological changes becomes apparent. The constant trauma of the muscular wall over the unyielding calculi is productive of degeneration and fibrous replacement of the essential tissues. The sac, because it is now merely fibrous tissue, contracts, gradually occluding its blood circulation, its means of vitality, slowly producing by a pressure atrophy a tissue such as in the body becomes calcified. Much emphasis has been placed in literature upon the infective element in etiology, but the insignificant part it plays in this process of calcification is reasonably obvious. In the example provided the calculi are of the mixed type, which may indicate that at some phase a degree of infection was present in the gall-bladder. At no time, however, was it sufficiently active to produce symptoms. Were bacterial infection at all a marked feature, such a stagnant, devitalized cyst, unable to evacuate its contents, would be extremely liable to become the nidus of an acute inflammatory process. Gall-stones with infective cholecystitis is the usual sequence. It is the rare exception in which the infection is nil or remains in abeyance and not a cholecystitis but a calcification results, where not infection but interference with function dominates the changing picture.

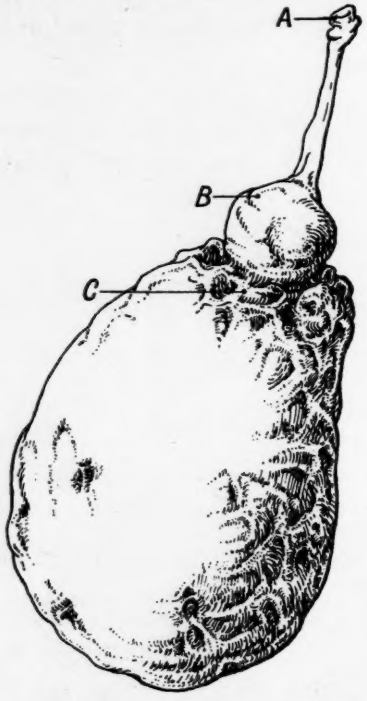

FIG. 109.—Drawing of Specimen 15 (2) j 2. A calcified gall-bladder with no calcification in the valvula spiralis. A, Cystic duct; B, Calculus impacted in cystic duct; C, Valvula spiralis.

As a further and probably more convincing proof of these etiological factors, the specimen 641·1 O.C. 2808 A, presented by Dr. R. F. Barkwell, 1888, in the Hunterian Museum, London, may be cited. In it there is no evidence of any sort to point to an infective element having been present. The gall-bladder is completely calcified, and its interior is fully occupied by four cholesterin stones.

Since it would seem to be true that by a contraction and pressure atrophy, calcification of the gall-bladder may result, it would also be reasonable to expect that a simple dilatation atrophy would result in a similar condition. Literature is void of help in this quest, but in the College of Surgeons' Museum, Edinburgh, a specimen of this condition is seen (*Fig.* 109).

Specimen 15 (2) j 2.—" From a woman, æt. 53, who died of carcinoma of the stomach. The gall-bladder projected about 7 cm. beyond the liver margin. The cystic duct was blocked by the only gall-stone which the gall-bladder contained. There was a history of a prolonged and severe attack of pain five years previously, but none of jaundice ".

The sequence of changes ending in such a large, dilated, and calcified gall-bladder appears to call for little question: the lone cholesterin gall-stone impacted in the cystic duct with the resulting mucocele; the consequent distention and loss of function leading to atrophy in the muscular strata, fibrous replacement, and finally calcification. Again, it is noteworthy that the calcification has taken place throughout the entire corpus and surrounding the gall-stone. Beyond the impacted stone the cystic duct has remained normal in structure. The valvula spiralis (Heisteri) also, though intracystic, has maintained sufficient function to thwart degeneration and has remained uncalcified. It is therefore apparent that the stress of the dilatation is the chief causal factor in the degeneration, since only where stress has overcome function has calcification resulted.

Passing from these two types which we choose to term the non-infective types of calcified gall-bladder, we turn to the infective types, where an inflammatory fibrosis has terminated in calcification.

Osler, in his paragraph on calcification of the gall-bladder, remarks that " calcification may be a termination of suppurative cholecystitis." As a sequence of events it is a tempting one to accept. It provides for such a degree of inflammation as is bound to result in degeneration of the organ, and also a reason for the extreme rarity of the condition, for a true suppurative cholecystitis must as seldom spontaneously subside as calcification of the gall-bladder is rare. It may, however, be mentioned that, though it may be rash to preclude at this stage in our knowledge of body calcifications such a rapid and acute tissue necrosis as a possible precursor to calcification, nowhere in the histories of cases is there any evidence to suggest such a sequence of events. Rather are calcifying gall-bladders peculiarly void of symptoms, and particularly of acute symptoms, till their later stages are reached, when, ceasing to be a vital tissue, the body requires to deal with them. Sternberg writes: " As the result of long-continued chronic inflammation, the gall-bladder often becomes smaller, and can as a consequence of suppurative processes shrink into a scarcely cherry-stone-sized, dense, fibrous, not rarely also calcified body, enclosed in pseudo-membrane." Kaufmann states that as the result of chronic catarrhal inflammation, sometimes without stones, " the thick, calloused, often entirely smooth wall can calcify, even ossify." In these last two statements is found what appears to be the truth regarding calcification in association with infective conditions of the gall-bladder. It is the termination of a chronic cholecystitis. Nowhere in the history of the infection does an inflammation accrue sufficient in its acuteness

to produce a tissue destruction, but merely a tissue degeneration and fibrous metaplasia. The inevitable progressive contraction of scar tissue follows, with the slow elimination of its vitality and the deposition of calcium salts within it. Such a gall-bladder may or may not contain gall-stones. Though the calcification may involve both the corpus and cystic duct, the latter is always patent and not obstructed by a calculus.

An illustration of this condition is seen in the Hunterian Museum, London, which we reproduce in actual size (*Fig.* 110).

Thus far we have confined our remarks to that type of calcified gall-bladder classified by Osler as "the true infiltration of the wall with lime salts, the so-called ossification." There is, however, another type which does not mean a calcification of the whole wall of the gall-bladder, and which Osler also mentions as "the incrustation of the mucosa with lime salts". An example of this condition is seen in the College of Surgeons' Museum, Edinburgh, 15 (2) j 3: "A gall-bladder distended and everted to show its coats, and on its inner surface a patchy deposition of lime salts". Here, though in a dried specimen, it is evident that this calcium deposit is merely in the mucosa, and that it may be shelled off in plaques, leaving the remainder of the wall of the gall-bladder intact. Fowler makes mention of yet another condition, "loose sand outlining the gall-bladder", an example of which is provided by Carman in the 1920 edition of his book. It would seem unnecessary to separate these two conditions. Both are depositions of calcium salts in the mucosa, and it is probable that the latter is merely an early stage in the development of the former. Etiologically we are dealing with a calcium deposition in these cases which cannot be explained as can the true infiltration of the wall, and neither histories nor specimens are available to assist us in elucidating the sequence of changes which lead to this selective calcification.

FIG. 110.—Specimen 2808. "A dried gall-bladder of very small size and having an extensive deposit of earthy matter like plates of bone in its coats. It is probable that previous to this deposit its coats were diseased. Presented by Sir William Blizard". The drawing represents in full size the bladder split longitudinally and opened out, so that the marked degree of contraction in its walls is apparent. No calculi were present.

We would therefore classify calcified gall-bladders after the manner of Osler: diffuse calcification of the gall-bladder wall and selective calcification of the gall-bladder mucosa, the former being divisible into those resulting from non-infective atrophies and those resulting from infective atrophies.

The production of symptoms in these cases appears to be brought about in a variety of ways. The non-infective varieties usually produce no symptoms and are only discovered when the abdomen is explored for some other reason. Where symptoms do develop, they appear late in life and result from a periglandular reaction—an attempt to shut off from the body tissues, by a fibrous hyperplasia and adhesion formation, what has become simply a stone, calculi fixed in a calcium matrix set in a calcified body. Again, where the large stagnant cavity remains open to direct infection from its duct, it may become suddenly the site of an acute abscess without any previous

indication of gall-bladder inflammation being present. Such was the case in the example recorded by Haldane. In the chronic infective types the symptoms are those of chronic cholecystitis or cholelithiasis, extending over a period varying from fifteen to thirty years.

It is unnecessary to dwell on the mere numerical rarity in the incidence of calcification of the gall-bladder, a rarity which has been amply proved by Fowler in his many communications both with röntgenological and clinical authorities; but some comment may be made as to the reasons why this rate of incidence should be so low. Firstly, it is not the usual thing to find perverted or overstrained function alone producing atrophic changes in the gall-bladder. Secondly, it is unusual to find infection of such a sac as this remaining so indolent that over a prolonged number of years it may exist, yet be compatible with normal function. Lastly, the calcified gall-bladder is most often so quiescent in its development, so unproductive of symptoms, that the existence of many must never be known or even suspected, and they are cast upon the rubbish heap of treasures whose only signpost is senile decay.

I am indebted to Mr. C. A. Moore, Bristol General Hospital, for permission to utilize his case in this article, and to Professor E. W. Hey Groves for his assistance in the collection of material and details. To Sir Arthur Keith, Hunterian Museum, London, and to Mr. Greig, College of Surgeons' Museum, Edinburgh, who placed the specimens in those museums at my service, I also express gratitude.

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Hunterian Museum, London.—Specimen 641·1, O.C. 2808 A. (Dr. R. F. Barkwell, 1888); Specimen 2808 (Sir William Blizard); Specimen 2806 (Dr. Goodhart, 1875); Specimen 2823 (E. Lloyd Bagshaw, Esq.); Specimen 2823 A (W. A. Carline, Esq., 1916).

College of Surgeons' Museum, Edinburgh.—Specimen 15 (2) j 2; Specimen 15 (2) j 3; Specimen 15 (2) j 4.

FRACTURES OF THE UPPER END OF THE FEMUR.

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"The deep slumber of a decided opinion" is always a dangerous form of repose for the scientific mind, and a contribution which awakens new interest and stimulates informed discussion upon any subject becomes thereby a thing of value. A recent article in this Journal[1] by George F. Stebbing is of this nature. It is original in its treatment, and raises several fresh points for discussion in the subject of fractures of the upper end of the femur. Two challenging assertions are made: firstly, that violence applied in the region of the great trochanter with a breaking force produces as its usual result a fracture of the acetabulum; and, secondly, that the common means by which fractures of the intertrochanteric type are produced is by indirect violence due to sudden pressure by body weight upon the head of the femur with the limb fixed—or, we may assume conversely, by sudden violence exerted along the line of the shaft of the femur, the body weight fixing the head in the acetabulum.

Fig. 111.—Application of forces to which fracture of intertrochanteric type is attributed by Mr. Stebbing.

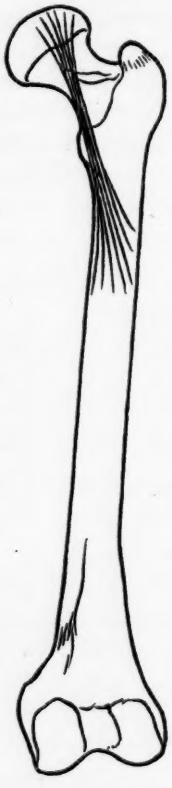


Fig. 112.—Lines of force in femur which indicate how intertrochanteric fracture is an improbable result of vertically applied forces.

The action to which Mr. Stebbing attributes the breaks through the trochanter (*Fig.* 111) would seem at first sight to be a reasonable result of the angulation of the bone at the junction of neck and shaft; a study of the divided bone and of skiagrams shows, however, that the main lines of support pass through from the shaft to the bearing-point on the head in a line, very slightly curved, on the inner side. This pillar of compact bone normally carries the body weight, and it is evident that quite an exceptional degree of violence applied vertically would be necessary to break it (*Fig.* 112).

The usual effects of the descent of the body weight upon the femoral head with undue force are well seen in some cases of 'lift' accidents, where a rapid descent is suddenly checked, and the force is transmitted upwards

FIG. 113.—Comminuted fracture from a blow over the great trochanter in the cadaver.

FIG. 114.—Intertrochanteric fracture with comminution. Blow over trochanter.

through the lower limbs. The common injuries which are produced in this manner, or by other falls from a height in which landing is made on the feet with the knees extended, are fractures of the os calcis, of the upper end of the tibia, of the condyles or the shaft of the femur, and occasionally of the occipital bone. Fractures through the trochanter may in rare instances be produced in this way, and most text-books accept the possibility of an occasional fracture in this region by a fall upon the feet or upon the knees. The suggestion, however, that all fractures in the intertrochanteric region are due to this indirect violence, and never due to direct violence applied to the trochanter, runs strongly contrary to clinical experience.

In an attempt to verify these counter-assertions some simple experiments were carried out upon the cadaver, all the subjects having been over 65 years of age at time of death, and upon dried femora. In some fractures, and particularly in regard to displacement, it is legitimate to argue that the conditions obtaining in life cannot be faithfully reproduced after

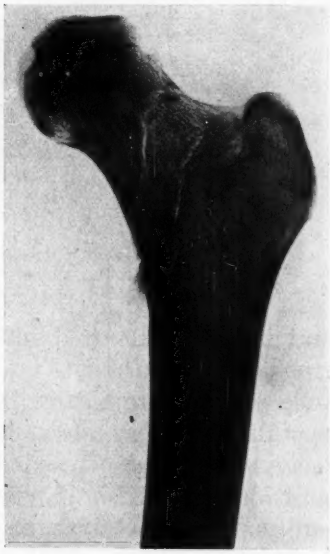


FIG. 115.—Vertical fracture produced in a dry femur by vertically applied forces.

death. On the other hand, when a statement is made that a direct blow upon the trochanter never breaks the femur but causes fracture through the acetabulum, and one finds in the cadaver that a direct blow on the trochanter does fracture the femur and does not fracture the acetabulum, one must hesitate to accept the dogmatic statement. When the experimental evidence is supported by the weight of many generations of observers, the balance in favour of the old-time view remains exceedingly strong.

The experiments carried out were of the following nature. In the first cadaver a direct blow was struck upon the trochanter, the limbs being adducted

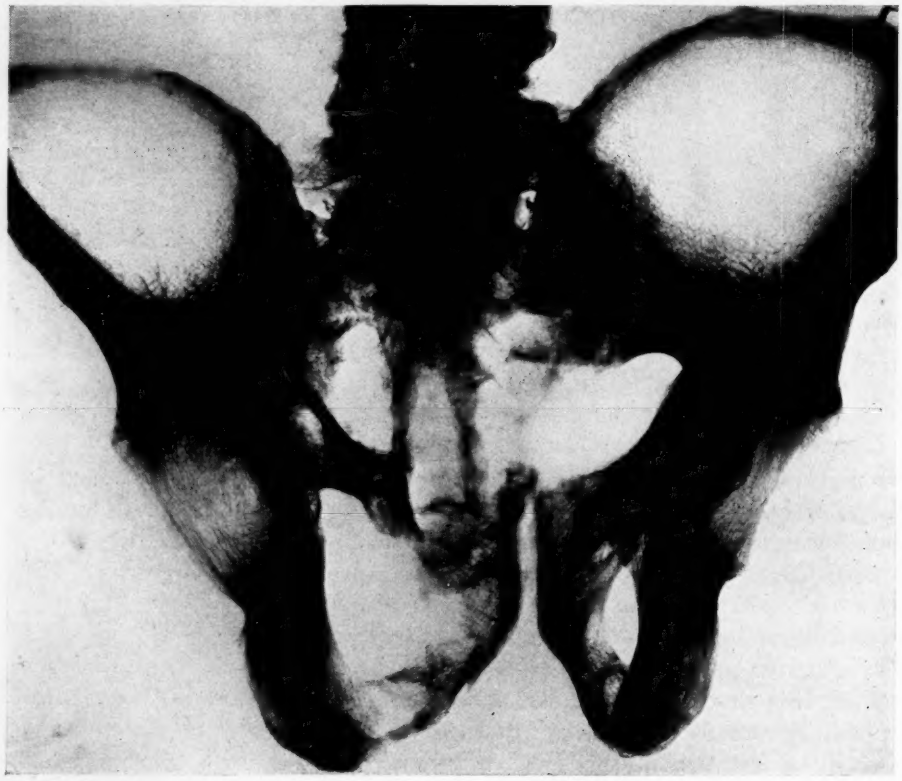


FIG. 116.—Multiple fractures of the pelvic girdle produced in the cadaver by slowly increased pressure over both trochanters. No acetabular injury.

and in slight external rotation. On one side a comminuted fracture of the trochanter occurred (*Fig.* 113), on the other a typical intertrochanteric fracture with comminution (*Fig.* 114). On the second body, in which, in the operative surgery class, bilateral excision of the knee had been performed, heavy blows were inflicted on the lower end of the shaft of the femur in all positions of the hip-joint. No effect was produced at the neck of the femur, or upon the acetabulum, and successive blows simply resulted in increasing local comminution at the point of impact of the mallet upon the lower end of the shaft. When the upper ends of the bones were exposed to be examined for damage, and when none was found, blows were directly applied to the

great trochanter, when comminution and fracture of the intertrochanteric type again resulted.

A dry femur was placed with the head on the ground and the shaft in vertical position. Blows were then applied to the condyles. In the absence of the shock-absorbing action of the acetabular bowl, slight flattening of the head took place, and finally an unusual fracture of the neck was produced in which the split ran approximately parallel to the pillar of compact bone (*Fig.* 115).

The third cadaver was placed in a large vice with parallel sides, and slowly increasing pressure applied over the two trochanters. A very great

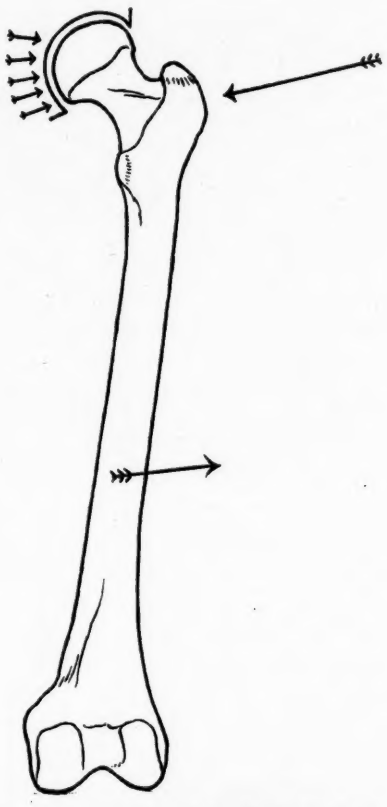


FIG. 117.—Leverage exerted by shaft as lower limb continues to fall when great trochanter is fixed against ground and head is fixed by body weight in acetabulum.

FIG. 117a.—Experimental reproduction of same.

degree of force was necessary before any sign of fracture occurred. Finally a definite crack was heard, and on dissecting away the soft parts it was found that fracture of the pelvis had taken place, as shown in the skiagram, in the horizontal pubic and the ischial rami (*Fig.* 116). Cracks were also produced in the ilia alongside the sacro-iliac synchondrosis. No trace of injury was present in the acetabula.

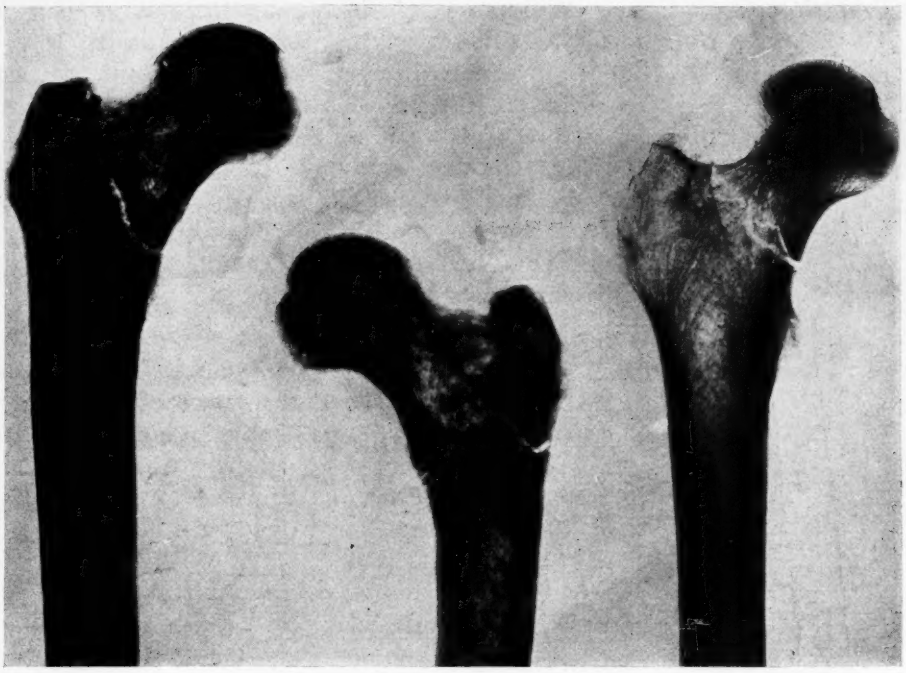
Another distribution of force was studied, which seems to be worthy of consideration. In the act of falling in a sideward direction the body tends

to sag at the pelvic level, and the trochanter and outstretched hand form the first points of contact with the ground. This is commonly seen in photographs of steeplechase and football falls, and is a well-remembered experience of those who have side-slipped on a frozen surface. In such a fall the upper end of the femur has for the moment two fixed points: the trochanter upon the ground, and the head of the femur fixed by body weight in the acetabulum. Powerful leverage is now exerted by the weight of the

FIG. 118.

FIG. 119.

FIG. 120.



FIGS. 121, 122, 123.

FIGS. 118–123.—Fractures through the base of the neck and the intertrochanteric region, produced by outward leverage of the shaft with the head and trochanter fixed.

still falling lower limb, with possibly the limb of the other side on the top of it, and this force, continuing to act, tends to break the femur at the trochanteric level. The type of leverage produced is shown in *Fig.* 117. If the upper end of a dry femur be held between two rigid flat surfaces which are in contact with the head and great trochanter, and leverage be applied by the shaft in an outward direction, exact reproduction of the common varieties of fractures through the trochanter follows (*Fig.* 117a). *Figs.* 118–123 show a series of bones to which violence was applied in this manner. In only one of these is there a horizontal fracture.

The mechanism of the various types of fracture in dispute is probably as follows. Fractures through the trochanters, and through the basal part of the neck, are due to direct violence applied to the great trochanter, whether the action be a direct shattering one or an outward leverage when the trochanter is fixed against the ground. As far as experimental conditions can be allowed for demonstration of fracture, these two mechanisms have been effective.

It is highly probable that the rarer fracture of the acetabulum occurs only in those cases in which force is applied truly through the capito-collar axis (*Fig.* 124). This force is usually of the nature of a heavy fall in which the area of bone immediately below the great trochanter strikes a raised object such as the kerb. Some buffer-crushing injuries are also of this type. In such cases an impacted fracture of the neck into the shaft may occur, or the

acetabulum may give way. Where a slowly increasing force is applied over both trochanters fracture of the pelvic girdle is usually produced.

FIG. 124.—Force applied through the capito-collar axis likely to break the acetabulum.

It has not been found possible in these few experiments to produce a fracture of the acetabulum by a direct blow, or by severe steady pressure over the great trochanter. It is conceded, however, that in very rare instances a fracture though the upper end of the femur which is atypical in line may be caused by the sudden descending weight of the body upon the head of the femur.

SUMMARY.

1. Fractures of the narrow part of the neck are due to an axial twist of the femur, an indirect violence.

2. Fractures of the broad part of the neck, and intertrochanteric fractures, are due to direct violence over the trochanter, or to outward leverage of the shaft when head and trochanter are fixed.

3. Fractures of the acetabulum are due to force transmitted directly along the capito-collar axis, by violence applied immediately below the great trochanter.

4. Fractures of the pelvic girdle may be produced by the application of slowly increasing, powerful pressure over the trochanters.

These observations were carried out in the Surgical Research Department, University of Edinburgh. I am indebted to Professor D. P. D. Wilkie for permission to use the material, and to Mr. Frank Pettigrew, chief technical assistant, for the X-ray photographs.

REFERENCE.

[1] STEBBING, G. F., *Brit. Jour. Surg.*, 1927, xv, 201.

CLEFT PALATE.

(Being a Hunterian Lecture delivered at the Royal College of Surgeons of England on Jan. 30, 1928)

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In the following paper a short account is given of cleft palate and its treatment. A new operative procedure, and its dangers and results to date, are described.

RESULTS OF OPERATION FOR CLEFT PALATE.

Of all the branches of surgery, that dealing with cleft palate is one of the most friendless. This I believe to be due to two very good reasons. Firstly, prolonged practice is necessary to enable the surgeon to attain sufficient skill in the type of operation chosen to assure a high percentage of successes in the closure of the cleft. Secondly, the results of any of the usually performed operations show such a very small percentage of good or perfect speakers that the surgeon requires an overdose of optimism in order to enable him to carry on.

From the standpoint of success in the closure of the cleft, the results are very encouraging. It is hardly fair, however, to expect the patient to show the same enthusiasm for a result in which the truly æsthetic value is more obvious to the surgeon than to himself. It is but cold comfort for him to possess a restored hard and soft palate and yet remain incapable of normal speech. Even so, he is almost always obliged to wear a dental appliance to supplement irregular or missing teeth; in which case why bother to repair the palate when practically the same effects are attainable by a skilful dentist? If the operation of gastro-enterostomy had to rely for its popularity upon such slender results as are obtained in cleft palate, the patients who would submit to such a procedure would be very few and far between. The present-day operations have, with certain modifications in technique, but not in principle, been practised for over a hundred years, and the time is now ripe to review the whole position and to draw up a profit and loss account. In this account the greatest item on the credit side must undoubtedly be the speech results. Union by first intention and considerations largely æsthetic should be given a very low value.

The aims in the operation of cleft palate are twofold: (1) To form a division between nasal and oral cavities whereby the patient is prevented from regurgitating food into the nose. The psychological effect plays a large part in the success of this. (2) To restore speech to normal, or, in the case of an infant, to enable it to speak properly in later life.

The first aim is achieved with a high percentage of successes in the hands of those skilled in this particular branch of surgery. There is no need to outline their methods, because they are well known and each practises

individual modifications, and on the whole the successes are of a fairly uniform high standard.

With regard to the second aim, the restoration of speech, the results are also fairly uniform ; but in this case they are uniformly poor. By that statement is meant the dreadful fact that, out of all the operations done for cleft palate, there are very few who ever have normal speech restored. Usually it is found, where a patient speaks well after operation, that he was also a good speaker beforehand. On the other hand, a patient who speaks badly before operation will usually also speak badly afterwards. A further striking fact is observed—that many sufferers from cleft palate in whom no operation has been done at all are surprisingly good speakers. I have in mind a professional man who wears an obturator for a completely failed repair which was attempted in early youth (*Fig.* 125). I have heard people express surprise when they were told of his having a cleft palate. Moreover, he can speak almost as well without his obturator as with it. I believe that in such cases the result is largely attained by training, very often self-training, and that those who speak well do so more by their having done this than by having any material anatomical advantage over others. The first essential in this training is fundamentally psychological, and, as in any other uphill fight, depends upon an impelling desire to overcome difficulties, stimulated either by ambition or a sense of shame. Where these impulses are absent, no training, operation, or other mechanical device will ever succeed. The second essential to good speech is physiological, and as far as possible anatomical, restoration of the palatal mechanism. We know sufficient of the results of the present-day operations for cleft palate to be able to say definitely that the second essential is rarely obtained. What, then, can surgery offer to the sufferer from cleft palate ? The answer is about a ten to one chance against his ever becoming a normal speaker, and the doubtful advantage of being able to deliver nasal speech with an intact hard and soft palate. The dental surgeon can do as much as this.

The object of this discussion, however, is not to decry the results of others but to see how they can be improved. Before tackling the problem, it is essential to have a clear idea of the anatomy of speech. Only that part directly concerned with the palate is relevant to this paper, and it alone will be considered in detail.

ANATOMY OF SPEECH.

During normal speech the soft palate is kept constantly in motion, being alternately drawn towards, or away from, the posterior pharyngeal wall. It forms part of a complicated muscular valve whose function is occlusion of the nasopharyngeal isthmus and the separation of the nasal and oral cavities. A cold in the nose frequently causes involuntary complete closure of this valve, and its effects are too well known to need any amplification. In normal speech, complete occlusion is necessary to enable clear pronunciation of all consonants except 'm,' 'n,' and 'ng'. Such efficiency is not so essential for the vowels. As the greater part of our language is made up of consonants it therefore follows that complete occlusion (cold in the nose) is fraught with much less disastrous effects upon speech than inability to occlude (cleft

palate). In cleft-palate speech the distinctness or otherwise will depend upon the relative amounts of sound projected through the mouth and nose. The nasopharyngeal valve is extremely active and moves with mercurial swiftness, and in very rapid speech must open and close many times in a second. It consists of two main parts, a palatal and a pharyngeal. The palatal part possesses two groups of muscles with opposing functions. One group, comprising the abductors, consequently opening the nasopharyngeal valve, consists of the palatopharyngei, the palatoglossi, and the tensores of the palate. The second, or adductor, group, closing the nasopharyngeal valve, is composed entirely of the levatores of the palate. Their action can be most clearly seen in a normal palate where 'Ah' is pronounced with the mouth wide open, a deep dimple forming on each side of the soft palate at the points of insertion of the muscles.

The pharyngeal part consists of an extremely complex muscle, the ultimate ramifications of which have not yet, to my knowledge, been adequately described. For this purpose it is sufficient to isolate those parts which have some practical bearing. The chief amongst these is the superior constrictor of the pharynx. In order to understand more fully the importance of this muscle it is necessary to diverge at this point. On examination of almost any unoperated case of cleft palate during the pronunciation of 'Ah' with the mouth wide open, a prominent ridge is seen running transversely across the posterior pharyngeal wall, to disappear into the upper reaches of the soft palate (*Fig.* 125). Passavant[1] was the first to describe this. If the bifid uvula be held aside during this movement, the outer end of the ridge is seen to enter the palate laterally to the muscular belly at the insertion of the levator on that side. The muscle appears to form part of the soft palate and, in contracting, it might be expected to exert some traction on this structure. This view of the structure of the muscle would appear to be morphologically compatible with what we know of its function as part of the nasopharyngeal valve or sphincter. Dissections of the pharynx of man and animals have shown this to be correct. With the help of my colleague, Mr. James Whillis, the superior constrictor has been shown to arise, not only from the usually described situations, but also from the palatal aponeurosis; a small fasciculus emanates from this situation to blend with the main portion of the muscle posteriorly (*Fig.* 126).

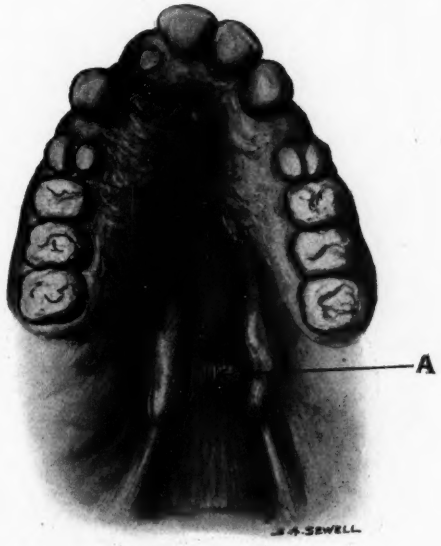

FIG. 125.—Cleft palate showing the ridge of Passavant (A).

In the resting state, the posterior part of the soft palate hangs down as a veil (*Figs.* 126, 127), parallel to, and not far removed from, the posterior pharyngeal wall. Between the two there is a narrow chink which is wide

from side to side, the cross-section area reaching a fairly large total. During speech this space is alternately obliterated and opened as mentioned before, but the muscles which take part in the movement make relatively small excursions, the width of the valve from side to side meaning that a slight

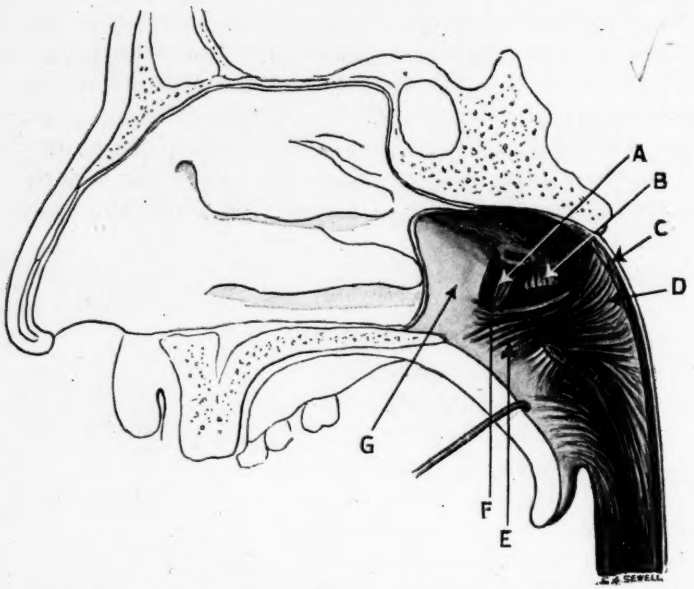

FIG. 126.—Dissection of normal nasopharynx. A, Tensor palati; B, Levator palati; C, Buccopharyngeal fascia; D, Superior constrictor; E, Fasciculus passing to soft palate; F, Fasciculus passing to internal pterygoid plate. G, Internal pterygoid plate.

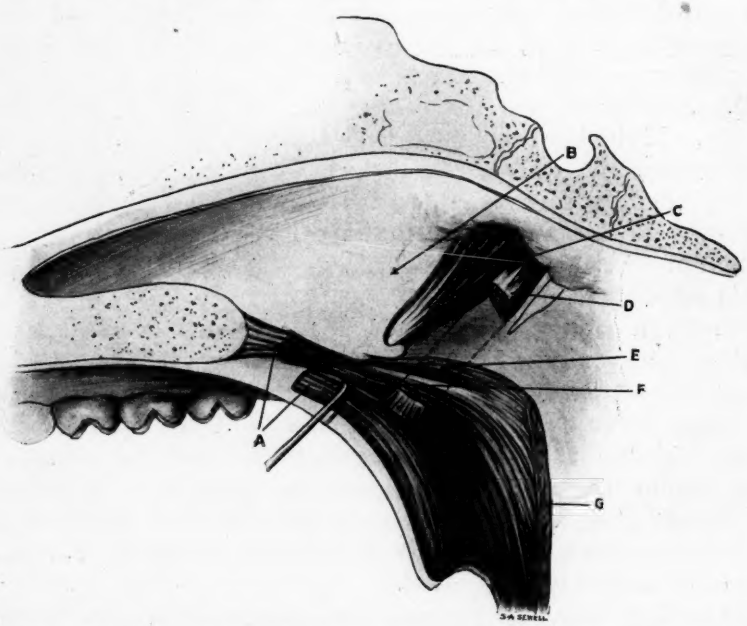

FIG. 127.—Dissection of nasopharynx of cow. A, Musculus uvulæ; B, Internal pterygoid plate; C, Tensor palati; D, Levator palati; E, Fasciculus passing to internal pterygoid plate; F, Fasciculus passing to soft palate; G, Superior constrictor.

alteration in the antero-posterior diameter makes an immense difference in the total cross-section area. This principle is applied in the case of the valves of the petrol engine, where a small linear movement of the valve allows of a large cross-section area for the inlet of petrol vapour or the outlet of exhaust gases. Roughly, the average maximum antero-posterior excursion of which the soft palate is capable is about 15 mm. (*Fig.* 128).

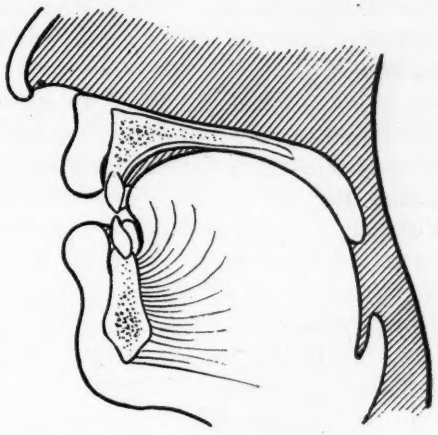

FIG. 128.—Tracing from skiagram. Normal palate: resting position.

The way in which this valve is operated is as follows. The superior constrictor muscle contracts and elevates the ridge of Passavant, thereby narrowing the nasopharynx antero-posteriorly and also from side to side. It remains in this state of contraction throughout speech, and only when speech ceases does it relax. The feeling of slow relaxation which normally occurs in the posterior pharyngeal wall on cessation of speech is readily recognizable by all who are conscious of their own muscular movements. Contraction of the superior constrictor and the formation of its transverse ridge are, then, probably for the purpose of forming a buffer against which the highly mobile soft palate can flap. The site of contact between palate and pharynx is at the level of the anterior arch of the atlas (*Fig.* 129). Once again, using the petrol engine as an example, the superior constrictor forms a valve seating for the upper surface of the soft palate. In order to appreciate to the full the principles of cleft-palate speech, great stress must be laid on the delicacy of the nasopharyngeal valve. As has been shown, the soft palate in the resting state is only about 15 mm. from the posterior pharyngeal wall, and its maximum linear movement cannot be greater than this. The width from side to side of the nasopharynx being considerably greater than this, it means that if the palate is unable to meet the posterior pharyngeal wall by so small a distance as 1 mm., a considerable cross-section area of pharyngeal airway is left open. The extraordinary delicacy required in this valve should be properly appreciated. Moreover, it shows that examination of the palate by looking into the mouth is futile, because not even the keenest eye

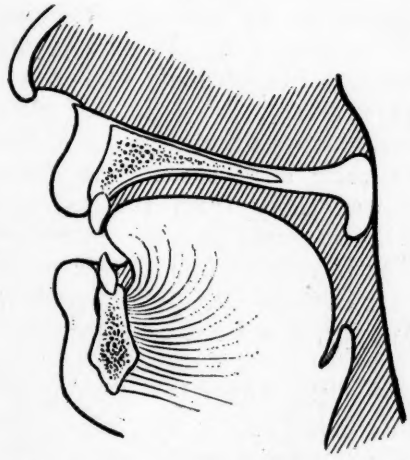

FIG. 129.—Tracing from skiagram. Normal palate: nasopharyngeal sphincter closed. This picture was made with the subject in the recumbent position, the nose being filled with bismuth emulsion. Note contact between soft palate and posterior pharyngeal wall.

could possibly appreciate a deficiency of a few millimetres between the palate and its valvular seating, when the essential parts are hidden from view. Most surgeons agree that many cases in which they have obtained apparently perfect anatomical results speak very badly, whilst others in which the anatomical result is apparently poor speak very well indeed. Let me repeat that the appearance of the anatomical results as seen from the mouth is no criterion of what is happening behind the palate, and as an observation is useless.

By the employment of an apparatus which I devised and demonstrated to the Royal Society of Medicine in June last, it was shown that normal speech depended upon a functionally competent nasopharyngeal valve, and that the worse the speech, after operation, the greater the deficiency in this respect. Further, it was shown in one particular case that noticeable cleft-palate speech persisted even when the total cross-section area of the deficiency of closure was as small as 48 sq. mm. Supposing the nasopharynx to be only 24 mm. in its transverse diameter (a gross underestimation of its size), this means that the palate in this particular case fails to meet the posterior pharyngeal wall by an average of 2 mm. The small size of the defect necessary to maintain noticeable cleft-palate speech is certainly very striking, and gives some indication of the extraordinary precision of the nasopharyngeal valve.

THE CAUSES OF CLEFT-PALATE SPEECH.

Cleft-palate speech results from two main causes: (1) An incompetent nasopharyngeal valve; (2) Incorrect speech habits designed to disguise this incompetence. I have proved this to my satisfaction by learning to mimic cleft-palate speech. That incompetence of the nasopharyngeal valve is not the sole cause is seen when the palate is paralysed as in diphtheria, the speech in this case being of an altogether different character from that of cleft palate. The difference lies in the fact that the subject of diphtheria has learnt normal speech; the tongue and lips go to their correct positions automatically; consequently the speech differs essentially from that of the cleft-palate speaker who has, in the course of his attempts to make conversation, developed incorrect anatomical habits. It is worth while at this point to examine some of these incorrect habits and see how the cleft-palate speaker, very often in spite of gross anatomical deformity, has been able to make himself understood.

Many cleft-palate speakers are able to say the letter 'k' with great facility. In the normal speaker this is done as shown in *Fig.* 130, where the pharyngeal valve is closed and the back of the tongue is forced against the roof of the mouth. Air is compressed behind the tongue, and its sudden release causes the explosive effort resulting in the sound 'k'. The cleft-palate speaker does this by throwing the tongue backwards and upwards towards the pharyngeal ridge of Passavant, and by completely blocking the cleft as shown in *Fig.* 131. Air is partially compressed in the space behind the tongue in sufficient quantity to make an explosive sound on release of the tongue. As this sound is not 'voiced', the quantity of air required is very small in amount. In the same way, many patients in the pronunciation

of the letter 's' learn to make the whistling sound, not between the teeth, but at the back of the pharynx between the tongue and the posterior pharyngeal wall.

The importance of understanding these abnormal tricks of speech is seen when one attempts to train a patient after operation. The letter 'k' has always previously been formed partly by a sort of cough and partly by releasing the tongue from its position in the cleft. After operation the patient is expected to close the nasopharyngeal valve, a function of which he has no previous experience, and while still keeping the valve closed to force air between the soft palate and the dorsum of the tongue. Acquisition of this simple feat is on a parallel with a man suddenly growing wings and being asked to fly. It can only be mastered by the practice of repeated trial and

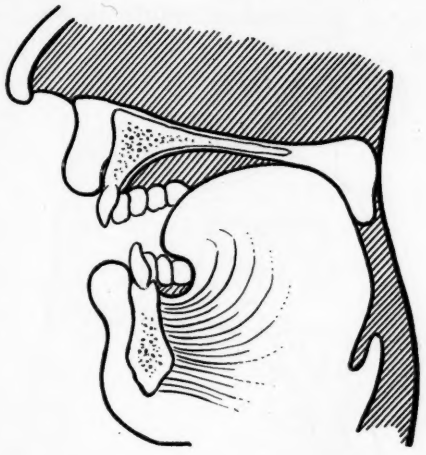

FIG. 130.—Tracing from skiagram. Normal palate during articulation of 'K.' Note contact between soft palate and posterior pharyngeal wall.

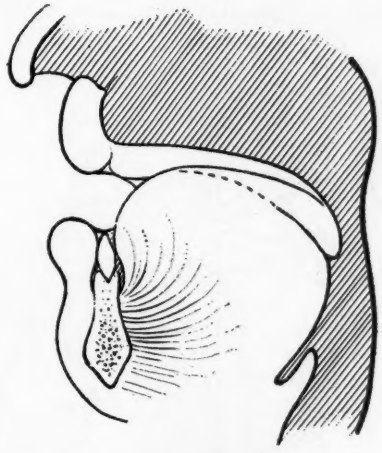

FIG. 131.—Tracing from skiagram. Unrepaired complete cleft of the palate during articulation of 'K.' No anatomical nasopharyngeal sphincter is seen.

error. In the same way the 's' has been made by whistling between the back of the tongue and the pharyngeal wall. This letter has to be shifted forwards to occupy the linguo-dental position. In some of my cases attempts have been made to study the best means of speech training after operation, and it is found that if the patient can once be made to produce a given sound by the proper method, repeated practice produces perfection. A useful way of making a patient acquire a letter, such as the 's', is to block up the nostrils with cotton-wool and then to demonstrate the correct positions for the tongue and teeth; a little practice soon brings the required result. The next step is to make the patient say the letter without the wool, closing, of course, the nasopharyngeal valve. The training of a cleft-palate speaker can only be likened to a normal speaker learning a new language, and in this connection one may be forgiven for quoting from the letters of Gertrude Bell, who, describing her efforts to learn Arabic, says "The pronunciation is past words, no western throat being constructed to form these extraordinary gutturals". If the patient, then, has acquired faulty methods of speech, he is faced with

the same difficulties as those mentioned by Miss Bell. Surely this is a great argument for performing any operation before speech has been acquired.

It is generally agreed that the results of the operation of median suture leave much to be desired, at any rate with regard to the all-important function of speech. The reasons for this are apparent in that this operation does not form a functional valve in the nasopharynx. It fails to do this because the soft palate is usually more rigid than normal, and is held in a position far removed from the pharyngeal wall. The two skiagrams shown (*Figs.* 132, 133) are from an adult of 45 years who recently underwent the Langenbeck-Fergusson type of operation. In the first the soft palate is shown in the resting position. In the second, the position of the soft palate is shown during the formation of the sound 'Ah'. It will be noted that the soft palate still remains at a considerable distance from the pharyngeal wall. This case is what is usually described as a very good anatomical result in that he

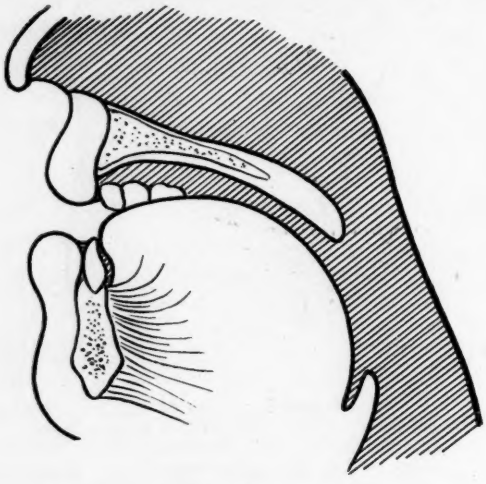

FIG. 132.—Tracing from skiagram. Case of cleft palate after Langenbeck-Fergusson operation; palate in resting position.

FIG. 133.—Tracing from skiagram, same case as *Fig.* 132, during pronunciation of 'Ah.' The soft palate, although mobile, does not reach the pharyngeal wall.

possesses perfect union and free movement of the palate. He is a very good speaker; but he was a very good speaker before operation and has really shown little more improvement than might be expected from the fitting of an obturator.

The speech tricks acquired by the subject with an open cleft have been mentioned, and the extreme value of the proximity of the tongue to the posterior pharyngeal wall has been shown. The way in which the tongue assists speech by blocking an open cleft has also been described. If, then, one substitutes for the cleft a repaired soft palate which cannot reach the pharyngeal wall, and also effectively prevents the tongue from so doing, speech will be worse, at any rate with regard to the posterior linguo-palatals. This is in accord with one's experience that very often, temporarily at any rate, speech is worse after operation.

ANATOMY OF THE DEFECT.

There are two great material changes in cleft palate: (1) *Defects in the soft parts*; (2) *Defects in the skull bones.*

1. **Defects in the Soft Parts.**—It seems to be generally agreed that the soft palate is shorter than in the normal, although this does not strike one in the accompanying drawing of a dissection of cleft palate (*Fig.* 134). One thing does, however, appear to be certain, namely, that the two halves of the cleft soft palate are not together equal to a whole normal palate. The musculature of the velum and nasopharynx shows no noticeable alteration from what can be seen in the dissections of the normal. On the other hand, in many cases

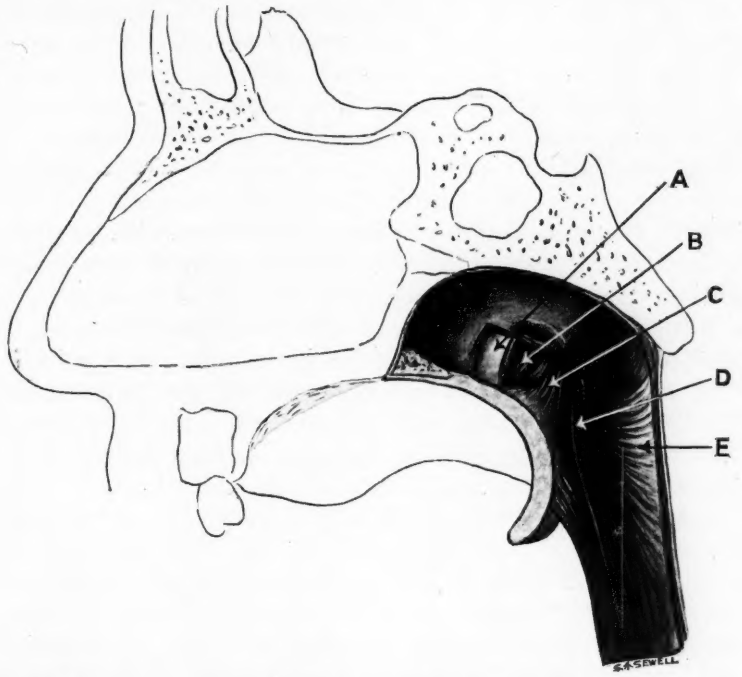

FIG. 134.—Dissection of nasopharynx in cleft palate. A, Internal pterygoid plate; B, Tensor palati; C, Levator palati; D, Salpingopharyngeus; E, Superior constrictor.

of unoperated clefts little or no movement can be seen in the muscles of the bifid palate or in the superior constrictor of the pharynx.

2. **Defects in the Skull Bones.**—Defects in the skull bones are more obvious than are those in the soft tissues. Firstly, in many cases of unilateral cleft, one finds gross asymmetry of the calvarium. The rough sketch shown in *Fig.* 135 was made from a specimen of a complete left-sided cleft in the possession of Dr. Victor Veau, of Paris, who kindly placed his material at my disposal for examination. It will be noted that the skull is markedly asymmetrical, and that the left parietal eminence is displaced considerably further backwards than its opposite fellow. This is not merely a freak case, but can be seen in many specimens of unilateral complete clefts, and surely is an indication that the effects of cleft palate are not merely local, but are

associated with widespread structural changes in other parts of the skull and perhaps even further afield.

When normal skulls are compared with those possessing cleft palates, changes, which are amenable to measurement, are readily recognizable. Measurements have been taken for the purpose of proving the existence of

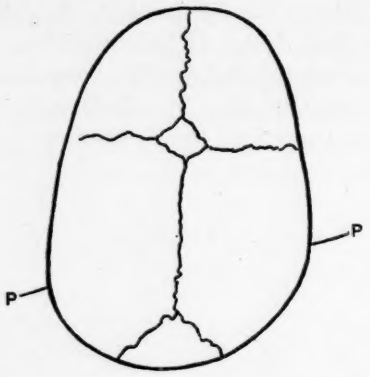

FIG. 135.—Diagram of skull bones from specimen of complete left-sided cleft, to show the backward displacement of the left parietal eminence (P). (*Dr. Veau.*)

bony defects in the nasopharynx. For comparison with normal skulls of about the same size, the distance between the glabella and occipital point, and the greatest transverse diameter, have been used in most cases; but in many the former distance has been replaced by that between the nasion and occipital point. This has been done because in many of the available specimens of cleft-palate skulls a large part of the calvarium had been removed.

CLEFT-PALATE SKULLS COMPARED WITH NORMAL.

	CLEFT PALATE (mm.)			NORMAL (mm.)		
	Nasion to Occipital Pt.	Trans. Diam.	Trans. Hamuli.	Nasion to Occipital Pt.	Trans. Diam.	Trans. Hamuli
Musée Dupuytren	180	153	38·5	176	148	35
" "	162	149	43	171	133	28
Veau 23	156	133·5	32	160	133	33
" 2	127	117	27	151	124·5	23
" 0	123	103	24	148	124	26
" 51	122	101	24·5	133·5	117	23·5
" —	121	102	31	120	92	21
" 10	117	111	31	112	90	18·5
" —	101	82	23	107	84	22
" 12	100	94	26	103	90	19
" 192 F	93·5	88	24·5	99	82·5	19·5
" —	93	83	25	96	82	17·5
	Glabella to Occipital Pt.	Trans. Diam.	Trans. Hamuli	Glabella to Occipital Pt.	Trans. Diam.	Trans. Hamuli
Nubian R.C.S. ..	175	128	41	178	149	34
Terat R.C.S. ..	187	150	42	190	147·5	36
D.U.C.M. ..	187	145	43·5	195·5	141·5	32·5
" " ..	169	125	39·5	180	141	30

The above measurements show that in cleft palate there is a definite bony increase in the transverse diameter of the nasopharynx. The increase is expressed only in terms of millimetres, and as such appears extraordinarily small in amount. But, as I have attempted to point out, the nasopharyngeal valve is of such precision and delicacy that even a minute change in its diameters will cause disastrous interference with its competence. How far these results are applicable to the living subject it is difficult to say. Moreover, the differences met with in cleft-palate subjects vary in the extreme.

One might expect that the patient with a cleft of the soft palate only would be easy to deal with and would give a good result. Experience shows that this is not so, the results even in this type of case being bad. I believe, but am unable to prove my belief, that there are bone changes in this type of case as in the others, and that very often the diameters of the nasopharynx in these cases are as much above normal as in many of the worse types of cleft, and that equally vigorous operative measures are as necessary in the one as in the other. The anatomical changes in the nasopharynx and palate are of the greatest importance in the consideration of repair. Firstly, we know that there is less than the normal amount of soft tissue, and that this has to be used to bridge an abnormally wide nasopharynx. Obviously, if the palate be sutured in the mid-line, its stretching from side to side will cause its shrinkage from before backwards. Secondly, each tendon of the tensor of the palate passes in a groove around the hamular process of the corresponding side to be inserted into the palatal aponeurosis. As the soft palate has been shortened by the mere process of suturing and stretching transversely, and as the widely separated hamular processes form the fixed bony points for the soft palate even after its separation from the hard, it follows that the palatal aponeurosis is bound to remain under tension, and consequently rigid, the upper surface of the soft palate being thereby prevented from reaching the pharyngeal wall. On theoretical grounds, suture of the soft palate without detachment from the hamular processes would seem to be one of the best ways of preventing the restoration of the nasopharyngeal valve. This, I believe, is one of the main reasons why the operations at present practised fail. At any rate, it probably accounts for one factor in failure, namely the immobility which often vitiates an otherwise apparently perfect repair.

THE SURGICAL PROBLEM.

The problem of the treatment of cleft palate is one in which surgery plays a very small but important part. Speech is essentially a material thing in that sounds are caused by vibrations in the air set up by vibrations in solid structures. In a musical instrument we can, by alterations in structure, vary the sound which will be emitted, but we do not expect to be able to produce musical sounds out of instruments which are structurally at fault. In the same way we cannot expect to restore normal speech to the cleft-palate sufferer unless he is supplied first with an instrument which is structurally and functionally sound. The greatest fault, common to all the usually practised operations, is the failure to restore a functional valve or sphincter. This, then, is the first fault to be remedied, and until this fault is rectified

perfect speech will never be attained. But, as has been shown, there is no operation at present in which a sphincter can be formed with certainty in every case. In fact, it is only in the most unusual cases that this happens, and with such infrequency that the result can only be regarded as fortuitous. If, then, a new pharyngeal valve is formed, the first step has been taken in the restoration of normal speech; but, even assuming the subject to be capable of using this valve, there are often other structural defects left which might detract to some extent from the quality of the speech emitted.

A NEW OPERATION.

The two-stage operation described below is based upon the preceding anatomical and physiological observations. It is founded upon the fact that the bony nasopharynx is increased in size, and that therefore any procedure which is content merely to restore a soft palate, however mobile, is bound to be a failure. Working on the principle that if the palate cannot be brought to the pharyngeal wall, the pharyngeal wall must be brought to the palate, the first stage of the operation was devised. The object of this stage is to narrow the nasopharynx laterally, and to produce a cushion of tissue on the posterior pharyngeal wall, imitating, but exaggerating greatly, that which is normally formed by the superior constrictor muscle, whereby a seating may be formed for the upper surface of the repaired soft palate. The operation is similar in principle to pyloroplasty, except that it is done the reverse way and for the purpose of producing the opposite effect.

First Stage.—The patient, lightly anæsthetized, is laid upon the back with a pillow beneath the shoulders and the head thrown well backwards. The mouth is held open with a gag. The pharynx is stimulated and the position of the ridge of Passavant noted. With a fine tenotome, the mucous membrane is incised transversely at the level of the ridge of Passavant, i,e., over the anterior arch of the atlas, and is then held open with fine sharp hooks (*Fig.* 136). Care is taken not to incise too deeply and thus open the loose areolar tissue between the buccopharyngeal and prevertebral layers of fascia. The buccopharyngeal fascia is relatively tough, and the superior constrictor muscle and its covering mucosa can be readily peeled or scraped from it by means of blunt dissection with a curved aneurysm needle. The dissection is carried on in this layer, upwards nearly as far as the base of the skull, downwards for a similar distance, and laterally a little beyond the ridge caused by the salpingopharyngeus muscle. During the whole of this part of the operation some resistance is felt to the dissection. If this resistance is not felt, it is probable that the point of the needle is in the loose retropharyngeal cellular tissue.

Using the aneurysm needle as a combined director and retractor, the incision is enlarged laterally right up to the salpingopharyngeus by the use of curved scissors (*Fig.* 137). Bleeding is very small in amount, there being little more than a slight ooze. The business of sewing up the incision in a vertical direction is now undertaken. I have had a small spring wire retractor made (*Fig.* 138) for the purpose of facilitating this procedure. The retractor holds the incision open in a vertical direction. Using very small curved

needles made on the Mayo principle, sutures of No. 0 catgut are inserted, commencing first at the upper and lower ends of the wound; the sutures are left long and may be used as retractors. It is important to take big bites of tissue on each side, as the sutures have a tendency to work out very quickly. The suturing is completed in the centre of the wound by joining together the two salpingopharyngeal folds, including a sufficient bite to obtain a firm hold

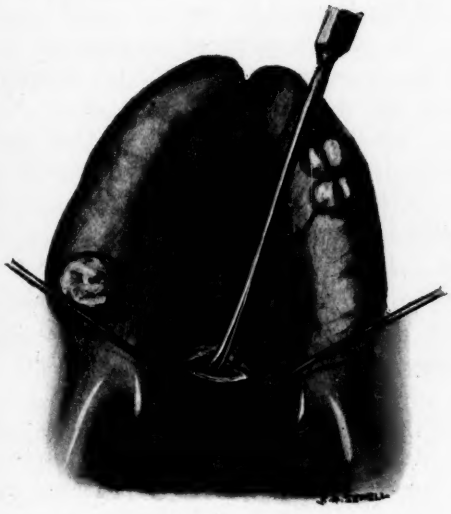

FIG. 136.—Separation of the superior constrictor from the buccopharyngeal fascia.

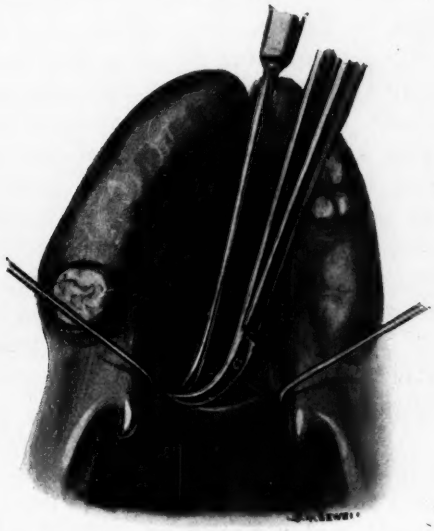

FIG. 137.—Extension of the incision as far as the salpingopharyngeus.

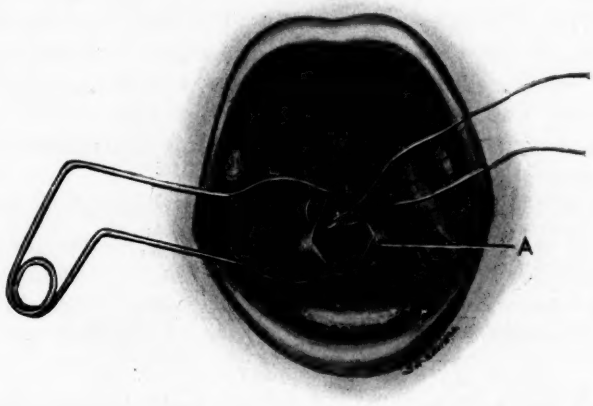

FIG. 138.—Suture of the wound in a vertical direction. A, Edge of salpingopharyngeal fold.

of the tough connective tissue which is intimately bound up with these. Usually a suture is passed through the posterior pillars of the fauces for the purpose of relieving the tension for a few days. At the end of the operation the halves of the soft palate are found to be much closer together, and the halves of the uvula may overlap. In fact one wonders how the patients are going to be able to swallow.

For the first few days the sutures hold and all appears to be going well.

There is little or no complaint of pain. About the fifth day the suture holding the salpingopharyngeal folds gives, and a small hole appears. Usually no further breaking-down occurs, and a permanent ridge on the posterior pharyngeal wall remains.

Second Stage.—The second stage of the operation is the repair of the palate, and is done after the wound of the first stage is healed and the tissues are once more in a settled state. This usually requires an interval of about three or four weeks. Either the Langenbeck-Fergusson or the Gillies-Fry method is suitable for this purpose, with the modification of division of the hamular processes or of the tendons of the tensores of the palate, thereby setting free the palatal aponeurosis, and so destroying the greatest bar to backward displacement of the palate. On theoretical grounds this step must be regarded as of the greatest importance, and in my experience it has certainly justified its performance. Whether in practice the simple Langenbeck-Fergusson or Gillies-Fry operation will suffice when combined with pharyngoplasty, remains to be seen. In seven cases I have done the Langenbeck-Fergusson, and in one the Gillies-Fry, operation. The large hole left between the hard and soft palates in the latter procedure offended my æsthetic sensibilities and drove me to do the Langenbeck-Fergusson as a routine.

Whatever the procedure adopted in the second stage, a few words of warning are necessary. If the first stage of pharyngoplasty has been successfully performed, repair of the palate completely blocks the nasopharynx for a matter of a few days until the sutures loosen. It is therefore extremely important that a careful watch be kept over the patient until recovery from the anæsthetic is complete. Ignorance of the possibility of this complication in one of my cases was nearly the cause of a fatality. Since then I have been in the habit of passing a silkworm-gut suture through the tongue for the purpose of traction. For the first few days breathing through the nose is often impossible, and, in order to accustom the patient to this before operation, in the last case of my series, the nostrils were blocked with cotton-wool for forty-eight hours beforehand.

Both stages of this operation can be done at one sitting, as in three of the cases recorded below. I do not think this is wise, because once the soft palate is repaired, the difficulties attendant upon any necessary subsequent interference in the nasopharynx are increased considerably. Moreover, in the two-stage operation one can study the effects of the first before proceeding to the second.

Dangers.—All operations, however trivial, have certain risks attached to them, and a certain mortality associated with these risks. The operative procedure which has been described is no exception to this rule. The dangers as known at present are mediastinitis, asphyxiation, and deafness.

Mediastinitis.—In my series of nine cases I have had one death which, in the light of further experience, was avoidable, and I am firmly of the opinion that similar occurrences will be few and far between. The fatal case referred to was that of a boy of 4 years, in whom was done the first stage or pharyngoplasty. He was the eighth of the series, and in the desire to obtain thorough separation of the superior constrictor muscle, it was not realized

that the buccopharyngeal fascia had been perforated, and that the dissection was being carried on in the loose retropharyngeal connective tissue. The operation was completed in the usual way. The child died thirty-six hours later from acetonæmia. At the autopsy a mild degree of retropharyngeal cellulitis was found, with a small purulent track passing down into the mediastinum. Although it is not thought that this child died from sepsis, it is preferable that the case should be labelled a direct operative death. If one is careful not to perforate the buccopharyngeal fascia, a repetition of this unfortunate complication should not occur. It is most strongly urged to anyone who might contemplate this operation to be most careful in the study of the anatomical facts, and, before attempting to perform it on the living body, to try it in the cadaver in order that a clear conception of the limits and texture of the buccopharyngeal fascia may be obtained. This is not an operation to be undertaken lightly, and before I myself first did it on the living subject, I had made many dissections of the cadaver.

Asphyxiation.—In discussing the treatment after the second stage, or repair of the palate, the danger of asphyxiation was mentioned. This is repeated here in order that it may be duly recorded on the debit side of the account.

Deafness.—Two of my patients have complained of deafness after the second stage of this operation. In one it was only transitory, in the other slight deafness still persists. Professor Grey Turner tells me that he has occasionally noticed the same complication after the Langenbeck-Fergusson operation.

Whether the dangers and disadvantages of the operative procedure recommended will in the end outweigh the advantages, time and experience alone will show. Full exposure of what are thought to be the disadvantages has been made not for the purpose of discouragement of others, but in order that others may help to put the operation in its proper place, either by its acceptance or rejection.

RESULTS OF THE OPERATION.

Case 1.—The first patient upon whom this new procedure was attempted was a miner, age 17 years. He had a cleft of the soft palate and about half of the hard. He had been operated on twice, at the ages of 3 years and 3½ years. Both operations were complete failures and the palate broke down. What was left of the soft palate was scarred and its mobility was to some extent interfered with.

First Operation (Feb. 8, 1927).—The patient was anæsthetized and a transverse incision was made through the mucous membrane about the level of the ridge of Passavant. The superior constrictor muscle was separated from the buccopharyngeal fascia for a distance of about an inch, but the salpingopharyngeal folds were not interfered with. The wound was then united in a vertical direction. At the end of the operation there was a distinct elevation of the posterior pharyngeal wall, and the two parts of the uvula were found to have been drawn much closer together. The whole procedure occupied over an hour, difficulties being encountered owing mainly to unsuitability of instruments and clumsiness of myself. The patient made an uninterrupted recovery from this stage, and no complaint of pain was made. The wound healed very well but not by first intention, a small area being left to granulate. At the end of three weeks there remained a small elevation of the posterior pharyngeal wall.

Second Operation (March 19, 1927).—The soft palate was separated from the hard and the hamular processes were divided. Repair was done according to the

method advocated by Gillies and Fry. Recovery from the operation was uneventful and later the patient was fitted with an obturator.

The soft palate, as might be expected from the pre-operative condition, was not very mobile. There was no evidence that the pharyngeal sphincter was competent. Speech remained of the nasal type. There were no facilities for speech training, but the parents expressed the opinion that speech was much improved. Of this I am not greatly convinced.

Case 2.—S. C., age 24, miner. Wide congenital cleft of the soft palate and all the hard palate as far as, but not including, the alveolar border. Speech very bad. The ridge of Passavant was not well developed, and there was poor movement in the levatores of the palate.

First Operation.—A small transverse incision was made through the pharyngeal mucosa. The superior constrictor muscle was separated and sutured in the vertical direction. No marked narrowing produced. The salpingopharyngeal folds were not sutured. After-progress was uneventful, and at the end of three weeks there was little to be seen of the original operation.

Second Operation (Aug. 19, 1927).—Repair of both hard and soft palates by Langenbeck-Fergusson method with division of the hamular processes. Short lateral incisions were extended around the last molar teeth. There was difficulty with regard to the suture of the hard palate, so the anterior extremity was left open. Recovery was uneventful except for the breaking-down of some of the hard-palate sutures. There was no evidence of a pharyngeal sphincter having been formed. Speech was improved, but not to any marked extent. Slight deafness was complained of shortly after the operation, and has persisted.

Case 3.—G. M., age 13. This girl had a cleft of about the posterior three-quarters of the soft palate. The nasopharynx appeared to be very large both anteroposteriorly and transversely. Speech was very bad indeed.

Operation (Sept. 30, 1927).—The mucosa of the pharynx was divided transversely and the superior constrictor separated, although not very freely. The wound was sutured vertically, with six stitches of catgut, the salpingopharyngei being included. Small lateral incisions were then made and the hamular process on each side was divided. After paring the edges, the soft palate was united in the middle line. Recovery was uninterrupted.

Speech in this girl was much improved as a result of the operation. The nasopharyngeal valve was restored, as evidenced by her ability to snort—that is, she can vibrate the soft palate against the posterior pharyngeal wall during the act of drawing mucus from the back of the nose into the mouth.

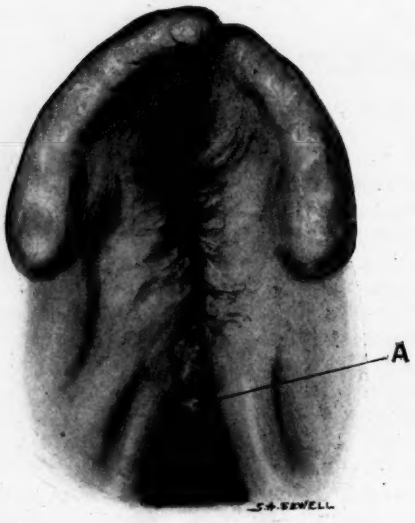

Fig. 139.—*Case* 4. Condition nine weeks after operation. A, Lower edge of pharyngeal ridge.

Case 4.—Dora S., age 36. Complete cleft of hard and soft palates, including the left side of the alveolar margin. Hare-lip repaired in childhood. Ridge of Passavant not well-developed; movement of levatores not very good. Speech poor.

First Operation (Aug. 31, 1927).—Formation of a ridge on the posterior pharyngeal wall by the usual method. No particular care was taken with the salpingopharyngeal folds. After the operation there was no complaint of pain and the wound healed in the usual manner. A well-marked ridge remained on the pharyngeal wall. The patient's friends noted an immediate improvement in speech, which was not, however, apparent to her medical attendants.

SECOND OPERATION (Oct. 7, 1927).—The hard and soft palates were repaired according to the Langenbeck-Fergusson method with the modification suggested.

Recovery was uninterrupted and the palate healed except for a small hole behind the alveolar margin (*Fig.* 139). The soft palate was fairly mobile. This patient-possesses a functional pharyngeal valve, as evidenced by her ability to snort. Her speech is improving greatly.

Case 5.—Miss N., age 26. Complete cleft of soft and hard palates, passing through alveolar margin on the left side, and associated hare-lip. The lip had been

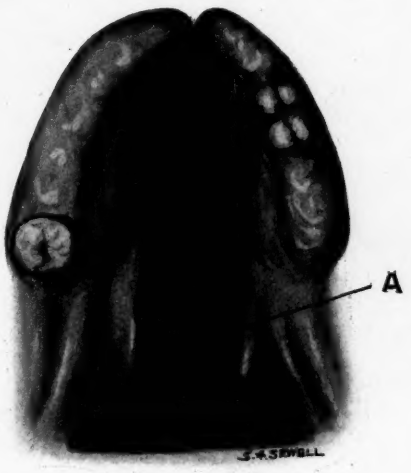

FIG. 140.—*Case* 5.—Before operation. A. Ridge of Passavant.

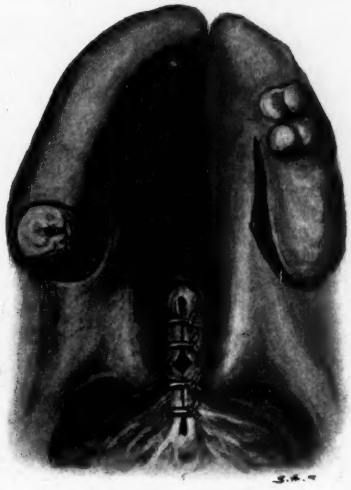

Fig. 141.—*Case* 5.—First stage completed. Lateral incisions made for Langenbeck-Fergusson repair.

FIG. 142.—*Case* 5.—Completion of second stage.

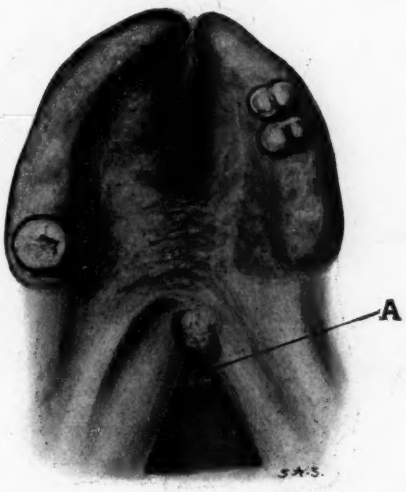

FIG. 143.—*Case* 5.—Seven weeks after operation. A, Lower edge of pharyngeal ridge.

repaired in childhood. At the ages of 11 years and 12 years she underwent operations for repair of the palate. Both were complete failures, and the resultant scarring was obvious. (Evidently extensive lateral incisions had been made). Speech was very poor indeed, but was improved by wearing a plate. The levatores of the palate were poorly represented. The ridge of Passavant was very prominent during

the pronunciation of 'Ah'; its ends could be seen to pass into the soft palate. It could be seen to remain in a state of contraction throughout the whole of speech.

OPERATION (Oct. 29, 1927) (*Figs.* 140, 141, 142).—A transverse incision was made through the pharyngeal mucosa and the superior constrictor separated from the buccopharyngeal fascia. The dissection was carried as far as the salpingo-pharyngeal muscles. Lateral incisions, as shown in the diagram, were made in the palate to relieve tension. The palate was then repaired by the Langenbeck-Fergusson method, the two stages being done at one sitting.

Later, the anterior part of the hard palate broke down. (The patient says she remembers putting her tongue through it.) (*Fig.* 143.) The soft palate was fairly mobile, and has remained so to the present. Examination shows the lower part of the newly-formed cushion projecting below the lower margin of the soft palate.

This patient now possesses a functionally competent nasopharyngeal valve. The evidence for this lies in two directions and is quite conclusive. Firstly, she is able to snort. Secondly, fluid poured into the nose can be held at the back without any escaping into the mouth. Unless the nasopharyngeal valve were competent neither of these feats would be possible. With regard to speech, this patient has shown great improvement in the short time which has elapsed since the operation. Many of her consonants are now emitted without nasal escape. The nasopharyngeal valve being now competent, there would appear to be no reason why the remaining sounds should not become perfect by practice.

Case 6.—Elsie F., age 15. Pre-operative condition showed a very wide cleft of the soft and hard palates nearly as far as the alveolar margin. There was wide separation of the alveolar margins, and very little tissue either in the hard or soft palate. Movement of the levator muscles was very poor, and no ridge of Passavant could be seen on the pharyngeal wall. Speech was absolutely unintelligible. This case was regarded as one of the worst possible to tackle.

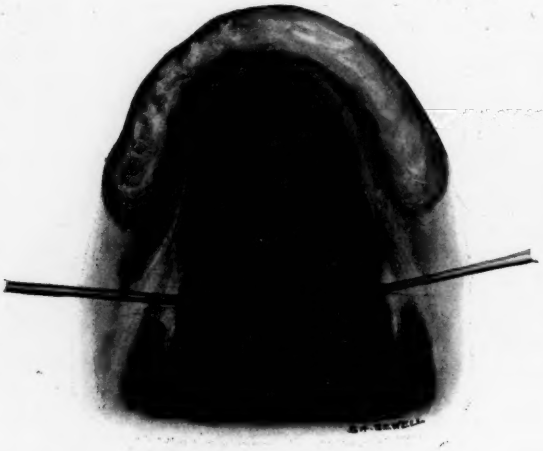

FIG. 144.—*Case* 6.—First stage preparatory to suture.

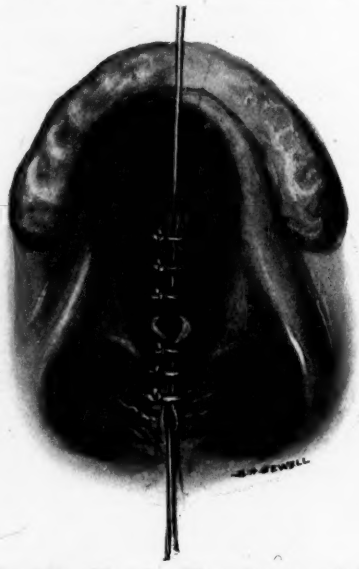

FIG. 145.—*Case* 6.—First stage completed. Note the approximation of the halves of the soft palate.

FIRST OPERATION (Oct. 29, 1927).—An extensive transverse incision was made in the pharynx, reaching as far as the salpingopharyngeal folds on each side. The superior constrictor was freely separated and then sutured in a vertical direction (*Figs.* 144, 145). A tension suture was passed through the faucial pillars. The two halves of the uvula almost touched at the finish.

Recovery after operation was uninterrupted and no complaint of pain was made. The wound appeared to break down about the fifth day, and it was expected that a complete failure would result. There was an intense hyperæmia of the pharyngeal

mucosa, but on this clearing up it was seen that a considerable pharyngeal cushion remained.

SECOND OPERATION (Nov. 25, 1927).—Repair by the Langenbeck-Fergusson method, care being taken to divide the hamular processes. Short lateral incisions passing around behind the teeth of both sides were made. The whole proceedings were very much embarrassed by alarming hæmorrhage, and it was at one time felt that continuation of the operation should be postponed. Fortunately it was found possible to complete the operation. It was thought that the hæmorrhage was in some way associated with the intense hyperæmia following the first stage. A silkworm suture through the tongue was necessary to prevent asphyxiation. Recovery was uninterrupted, except for the extreme discomfort associated with inability to breathe through the nose. A hole, as depicted in *Fig.* 146, was present fifteen days after the operation.

FIG. 146.—*Case* 6. Showing presence of hole fifteen days after operation.

It is of interest to note that during this girl's stay in hospital after the second stage, the other patients in the ward used to complain of being kept awake by her snoring.

The soft palate in this case, as might be expected, was very rigid, but that a functional valve was formed was evidenced by her ability to snort. Speech as yet shows little improvement.

Case 7.—Norman M., age 9. Presented a cleft of soft and hard palates as far as the alveolar margin (*Fig.* 147). A cleft, passing through the margin, had closed in the process of growth. The ridge of Passavant was poorly represented and movement in the levatores of the palate bad. There was a double hare-lip. Speech was unintelligible.

FIG. 147.—*Case* 7. Before operation.

FIG. 148.—*Case* 7. Condition five weeks after second operation.

FIRST OPERATION (Aug. 11, 1927).—1*st Stage:* A very inadequate pharyngeal plastic operation was done. The salpingopharyngeal folds were not sutured. Very little in the way of pharyngeal cushion remained after healing of the wound. The hare-lip was also repaired.

SECOND OPERATION (Nov. 6, 1927). *1st and 2nd stages:* A long transverse incision was made in the pharynx, and the salpingopharyngeal folds were divided. The wound was sutured vertically. The operation was carried out in such a way as to obviate any possibility of error in the formation of a pharyngeal cushion. The palate was then repaired by the Langenbeck-Fergusson method.

This was the first case in which it was realized that care must be taken to prevent asphyxiation before recovery from the anæsthetic. Recovery from the operation was uninterrupted. A small hole formed at the junction of the hard and soft palates, and one of the sutures in the uvula broke away, leaving the latter incompletely repaired (*Fig.* 148).

The soft palate was left relatively immobile, but the patient in spite of this was able to snort. Speech as yet shows little improvement.

Case 8.—John B., age 4. This was the case, the details of which are reported on p. 140, in which death followed thirty-six hours after performance of the first stage.

Case 9.—Wm. S., age 45. This was a complete cleft of the hard and soft palates as illustrated in the picture (*Fig.* 149). The cleft was not wide and the palatal arch was high. The ridge of Passavant was well developed and ran on each side into a wide lateral recess. The soft palate was mobile. Intelligence was rather below the average. Speech was in that group next to the worst, and was almost unintelligible.

FIRST OPERATION (Dec. 5, 1927).—A transverse incision was made in the superior

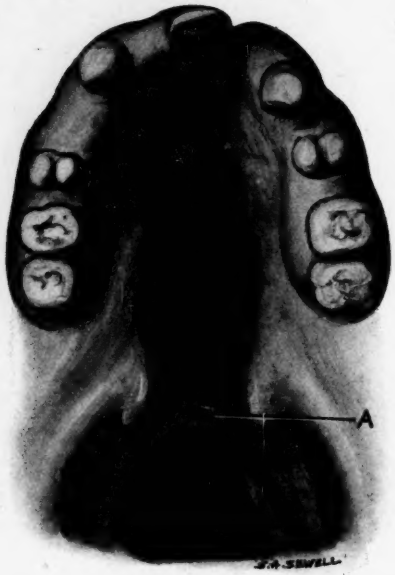

FIG. 149.—*Case* 9. Six days after the first stage. A is the artificial pharyngeal ridge, which has given a little in the centre. This ridge was examined six months later and no material change was noted.

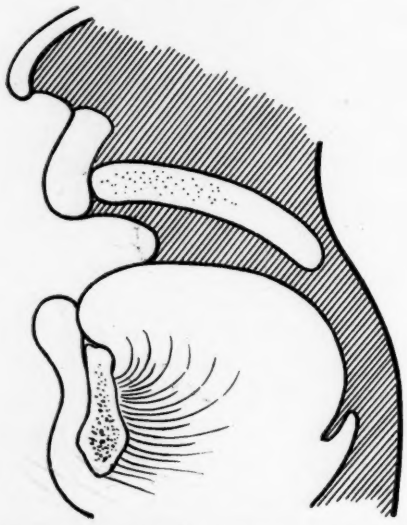

FIG. 150.—Tracing from skiagram of *Case* 9. Palate in resting position. After repair by combined Langenbeck-Fergusson operation and pharyngoplasty.

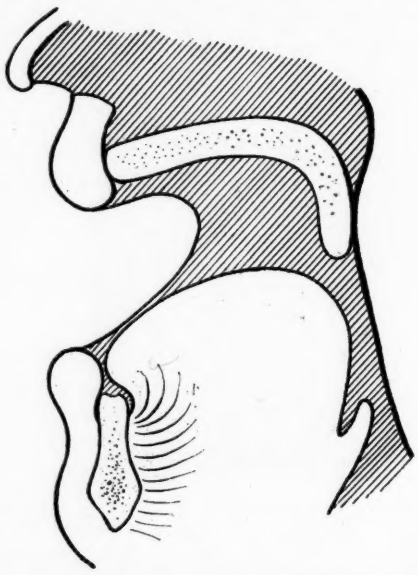

FIG. 151.—Tracing from skiagram of *Case* 9, after operation and during pronunciation of 'Ah.' The soft palate meets the posterior pharyngeal wall.

constrictor and the wound was closed vertically in the usual manner. Care was taken to suture the salpingopharyngeal folds, and one tension suture was fixed in the faucial pillars. At the end of six days the wound was found to have given a little in the centre; thereafter it remained stationary (*Fig.* 149).

SECOND OPERATION (Jan. 9, 1928).—The hard and soft palates were repaired by the Langenbeck-Fergusson method, the hamular processes being divided. Once again a suture through the tongue was necessary during recovery from the anæsthetic. A hole appeared at the junction of the hard and soft palates, and is now rapidly closing. A competent valve was formed, as proved by the ability to snort. Further evidence in this case lies in the fact that the pharyngeal cushion was made lower down than is the usual practice, and the lower edge of the soft palate can be seen to come in contact with this when he says 'Ah'. Skiagrams taken in the resting position of the soft palate and during the pronunciation of 'Ah' show convincing evidence of the competence of the valve (*Figs.* 150, 151). Speech at first was worse after the operation; but, even in the short time which has elapsed, improvement is beginning.*

In six of the cases mentioned above the ability of the patient to snort has been used as a proof of the fact that the soft palate comes in contact with the pharyngeal wall. To obviate any possibility of this being mistaken for stertor, the patient is always instructed to protrude the tongue and then grip it between the lips and teeth before applying the test. Many cases of cleft palate after the Langenbeck-Fergusson operation have been examined. Only one was able to snort: she was one of the rare cases of perfect speech result. The cleft was of the soft palate only.

CONCLUSIONS.

An attempt has been made to stress the importance of the formation of a pharyngeal valve in the sufferer from cleft palate, and it is believed to be impossible to restore normal speech unless this is first done. By the method described and applied in eight cases (excluding one death) this has been accomplished in six. In the first two cases the attempt failed for the good reason that the technique of the procedure was not fully understood and the anatomical points of importance could not be anticipated. Practice of the method has assured increased speed and certainty in the attainment of the result, and it is felt that future cases will be approached with a confidence that, at any rate in a large percentage, a functional valve will be formed. That this result cannot fail to reflect itself in improved speech must be obvious to all who have made a study of the subject.

In the discussion of the results of the procedure recommended, little mention has been made of the effects upon speech because of the short period of time which has elapsed since the earliest of the cases was submitted to operation. But there is abundant evidence, even at this early date, to make one sanguine of the issue. No claim is made to have found the solution of the problem in cleft palate, because all the surgeon can do is to provide a

* As mentioned above, this patient was able to snort after repair of the palate. He maintained this faculty for some weeks, when it gradually disappeared. Its loss was due entirely to contraction of scar tissue around the hole at the junction of the hard and soft palates. This gradually pulled the palate away from the posterior pharyngeal wall, leaving the artificial ridge completely exposed. There would appear to be no doubt as to the permanence of the ridge, which has not changed since the condition depicted in *Fig.* 149.

physiological palatal mechanism as a basis for the subsequent acquisition of speech. Perseverance, ambition, and shame are not yet included in the list of conditions amenable to plastic surgery, and so much depends on these abstract qualities, together with the intelligent guidance of a good teacher, that any claim to infallibility would be a claim to the impossible. Dr. Victor Veau[2] rightly asks, "Shall we ever know how to operate upon a cleft palate with as much security as upon inguinal hernia?"

The number of cases recorded is small, and all are of recent date. None of them was specially chosen for the purpose of swelling statistical columns; in fact, as a collection of cases upon which to attempt a new operation, they are as bad a lot as could be fairly expected. Two of them had had operations in earlier life (*Cases* 1 and 5), with consequent scarring. One had a wide cleft which might well have been adjudged inoperable (*Case* 6). In two (*Cases* 6 and 7) the muscle of the palate and pharynx was poorly represented and almost immobile even before operation, a fact which reflected itself in a relatively immobile palate afterwards; yet both of these cases were able to snort.

In conclusion a few questions arise in connection with the treatment suggested.

1. Will the cushion formed on the pharyngeal wall persist? Time and experience alone will provide the answer to this. In the meantime it may be stated that, so far as human observation can determine, there is no change in this respect over a period of months.

2. Is the method applicable to infants? The only child on whom the operation was essayed died. There appears to be no reason why, with the experience gained, the extension of the method to infants should not be attempted.

3. To what other uses may it be applied? There are large numbers of cases who have submitted to the Langenbeck-Fergusson procedure with disappointing results. The difficulties in the way of performing pharyngoplasty after a Langenbeck-Fergusson operation are not insuperable, and it is intended to try the method on the first opportunity.

4. With regard to adenoids. In none of the above cases was it necessary to consider this question, but it is obviously impossible to perform a reasonably neat and deliberate operation unless adenoids are first removed.

It gives me great pleasure to acknowledge the assistance I have received from many sources. Sir Arthur Keith and Dr. Victor Veau have kindly placed specimens at my disposal. Mr. James Whillis, Demonstrator of Anatomy in the Durham University College of Medicine, has assisted me to elucidate points in the superior constrictor of the pharynx. Professor R. B. Green has been generous in his supply of material and advice. To Professor G. Grey Turner I tender my grateful thanks for advice and criticism, and for allowing me to examine his cases. I am deeply indebted to Mr. T. W. Moles, M.A., for the time and patience he has spent in the speech-training of my cases.

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SPECIAL ARTICLES ON SURGICAL TECHNIQUE.

DIRECT SKELETAL TRACTION IN THE TREATMENT OF FRACTURES.

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It is now a generation ago since Codivilla first suggested the application of extending force to the fractured limb by means of a nail which was driven into the distal fragment of bone. This method, having been elaborated by Steinmann, was quickly adopted in Germany, Switzerland, and Holland, and more slowly in other countries. It has never been of general application in this country, although at the close of the War period, and especially under the influence of Pearson, it was used more frequently than any other method in the treatment of open fractures of the femur.

GENERAL PRINCIPLES.

There are two main advantages of skeletal traction in the treatment of fractures. First, it is by far the most efficient and reliable method for reducing shortening, or overlapping, especially if this has been of some duration. I would go so far as to say that, provided bony union of a fracture has not taken place, there is no malposition of a broken long bone which cannot be fully corrected or over-corrected by adequate skeletal traction, in regard to the full length and proper axis of the limb. Fractured femurs with three to five inches of shortening can be restored to full length, usually within a few days. And further, bad cases of mal-union can be similarly corrected if a preliminary osteotomy be done. In the second place, skeletal traction leaves the whole of the limb and the joints free for dressing of wounds, massage, and joint movements. This is of very great value in septic or open fractures, and in all cases when union is slow and the treatment necessarily prolonged.

Appliances.—Before describing the technique which I have found to be the most satisfactory, it may be worth while to discuss very briefly the different appliances which have been used by various workers for skeletal traction. These are represented by the following list: Transfixion pin; nails driven into the sides of the bone; ice-tongs calipers; horseshoe clamp with screws; os-calcis stirrup; silk or wire loops.

The *transfixion pin*, which goes right through the bone, I have always regarded as the best of all these devices, and after trying all the others I have come back to this, as the one which by reason of its reliability and simplicity

is the most satisfactory. The objection which has been urged against it is the liability to produce septic sinuses leading down to the bone. If due precautions are used, this drawback is rarely encountered, and an occasional sinus can easily be cured by simple curetting.

Nails driven into the side of the bone have nothing to recommend them. They are merely an improvised method used when the proper transfixion pin or tongs are not at hand. It is clear that two nails driven into the femoral condyles, for instance, cannot support a big traction force and will soon be dragged out of line by leverage.

The *ice-tongs caliper* is perhaps the method most widely used by those who employ skeletal traction, and it has been designed in a number of different shapes in order to ensure or to prevent the penetration of the outer shell of the bone by the points of the caliper. It is claimed for the caliper that it is superior to the transfixion pin, because it only holds the outer shell of bone and therefore does not cause a chronic sinus after its removal. But it is certainly more difficult to use for bad cases where a big traction force is necessary. Its correct adjustment in order to get the right degree of bone penetration is a difficulty, and this adjustment is liable to become disturbed during prolonged treatment. But this is perhaps a matter of personal predilection rather than one which involves any vital principle. Certainly the caliper requires to be supervised by nurses who are accustomed to its use, whereas the transfixion pin is practically fool-proof.

The various types of *horseshoe clamps* are open to exactly the same criticism as the calipers, and therefore no more need be said about them.

The *stirrup*, invented by Finochietto, which passes over the upper surface of the calcaneum between the tendo Achillis and the ankle, has the advantage of not requiring any bone penetration. But for this same reason, as it only goes through the soft parts, it is liable to slight movements, and this causes it to be much more painful than the rigidly placed transfixion pin.

Silk or wire suture loops have been described and used, chiefly for traction of the olecranon and the calcaneum. They are both neat and effective, but they do not present any real advantage over the transfixion pin, and if used for the heel bone they have the drawback of causing a wound on the plantar surface of the heel which may be troublesome.

PIN TRACTION APPLIED TO THE FEMUR.

It is not necessary to discuss the indications for skeletal traction of the fractured femur. The majority of surgeons will agree that it is indicated for all those cases neglected or insufficiently treated at the time of injury in which gross displacement has occurred with much overlapping and shortening. Also for cases of a later date where actual mal-union in faulty position has taken place. There is no general agreement, however, as to the use of this method in cases of recent fracture.

The apparatus—pin, handle, and traction stirrup—are made in different forms, but these varieties are of no essential importance. For the femur I use a pin 6 in. long and $\frac{3}{16}$ in. thick, with a bradawl point at one end and a flat bearing surface at the other which fits into a handle (*Fig.* 152). The patient

is always given a general anæsthetic, not only for the transfixion, but because the most important act of traction is the pull made upon the limb immediately afterwards, and for this full muscle relaxation is necessary. No doubt spinal anæsthesia would achieve the same object, but I have not employed this method myself.

The patient being anæsthetized and the skin prepared, the pin held in the handle is thrust into the soft parts without making a special incision for it, the integument being strained upwards towards the fracture whilst the transfixion takes place. The object of the latter detail is to lessen the tension on the skin when traction is applied; in the same way the pin, when it has gone through the bone, is made to emerge through the skin on the other side of the limb, the skin being pulled upwards. The site of transfixion should not be through the condyles, but well above them, through the lower part of the femoral shaft (*Fig.* 153, B). This is for two reasons: the cancellous tissue of the condyles is soft and gives way before the pressure of the pin, whereas the dense shaft will allow a big traction force without the pin working loose; and further, the high transfixion site avoids all danger of wounding the synovial sac of the knee-joint.

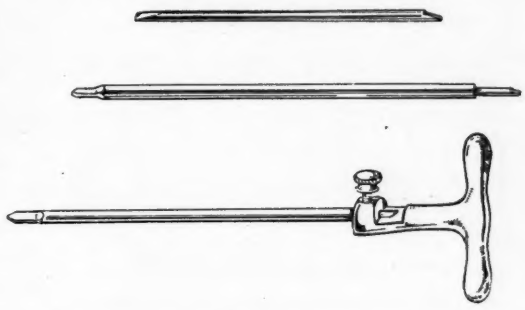


FIG. 152.—Transfixion pins and handle ($\frac{1}{3}$ actual size). (*From the author's "Modern Methods of Treating Fractures," John Wright & Sons Ltd., Bristol.*)

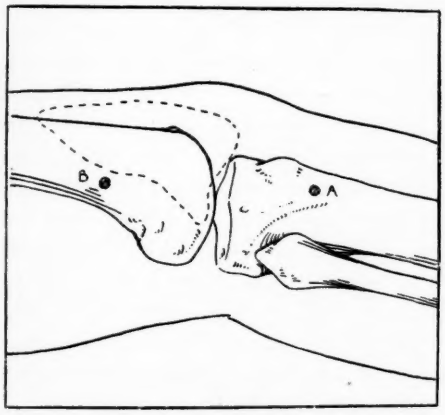


FIG. 153.—B and A, Points of transfixion of femur and tibia. Dotted lines show the limit of synovial membrane. (*From "Modern Methods of Treating Fractures".*)

The limb having been transfixed, there is about 1 or $1\frac{1}{2}$ in. of bare pin projecting on each side. This is covered with rubber tubing, which gives a non-slipping grip to the metal tractor, and also serves to protect the sharp ends of the pin, preventing them from scratching the patient or the nurse. Some layers of dressing are applied, with a bandage, and then the metal tractor shaped like a U is put on and kept in place by further dressing and bandage. The metal tractor has two small pulley wheels on its transverse bar, which now lies exposed below the knee-joint (*Figs.* 154, 155).

The transfixion having been accomplished and the tractor put in position, full manual traction is applied to the limb, and if any faulty union has already taken place this is broken down. The patient is put in a bed provided with a metal overhead bar to which the limb is slung, the knee being flexed to about 120°. The upright member of the overhead beam is provided with a

double pulley which can be adjusted at any height. A cord is tied to the fixed upright and passed alternately round each of the pulleys, two at the bed-foot and two at the knee, in the way shown in *Fig.* 155; finally, as it drops from the second pulley at the fixed upright it has the weight attached to it. This weight will vary from 5 to 15 lb. according to the type of patient and of fracture, giving with the fourfold pulley multiplication a traction force of 20 to 60 lb. The smaller weights are sufficient for fractures of recent origin or for small thin patients, whilst the larger ones are needed for old fractures in muscular subjects. In any case it is much better to begin with too big than too small a weight, because then the reposition of the overlapping fragments is more quickly accomplished, and directly this is done the weight can be diminished. The reduction of the weight is most conveniently done by altering the attachment of the cord, so as to use only 3 or 2 or 1 pulley. Generally the weight is thus reduced by one quarter at the end of one week, and by one half at the end of three weeks. If enough weight is used, then full reduction, and over-reduction, takes place; the fractured ends become disengaged from each other and

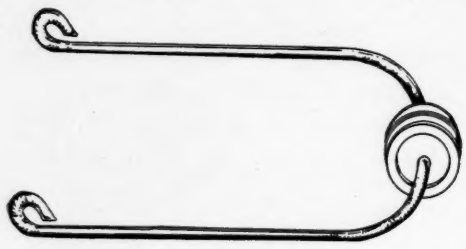


FIG. 154.—U metal tractor with pulley wheels.

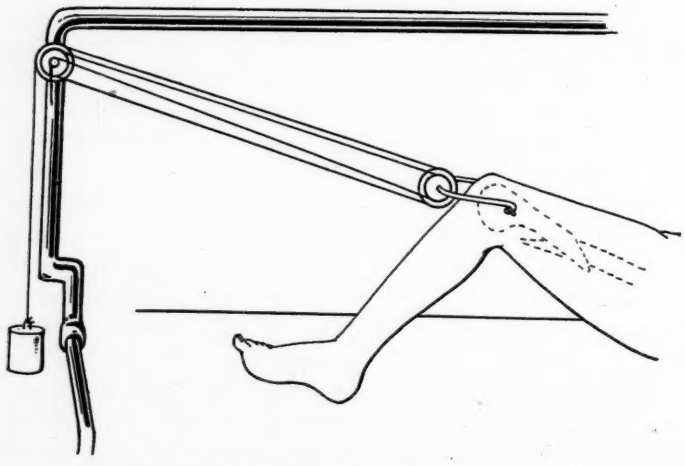


FIG. 155.—Multiplying pulleys. The pulleys are here drawn different sizes in order to show all four cords. Actually they are both the same size. (*From the author's article on 'Fractures of the Long Bones', in the "Index of Treatment", John Wright & Sons Ltd., Bristol.*)

from soft tissues, and can then be slowly allowed to assume the correct apposition.

In using these big weights efficient counter-extension is very important. The foot of the bed should be raised on 12-in. blocks and a padded perineal band passed round each thigh and tied to the corresponding upper post of the bed.

Usually the pin traction can be discontinued at the end of four to six weeks, and then its place may be taken by adhesive plaster traction or the use of a walking caliper. In the above description I have only spoken of the limb as being slung in a semi-flexed position of the hip and knee to an overhead beam. This is undoubtedly the technique which gives the traction force the fullest efficiency. But after a few days, before the end of the first week, the leg will have come to be of full length, and it is convenient then to apply a Thomas splint with a hinged leg-piece and foot-piece as a rest for the whole limb, without interfering with the traction.

Quite apart from special complications, pin traction of the fractured femur requires to be modified for two special positions of the fracture. In those high up, through the trochanters, it is essential to secure full abduction, and this can be done most conveniently by applying the perineal counter-extension to the sound side only, thus drawing up that side of the pelvis, whilst the broken limb is swung outwards to the side of the bed. In fractures of the lower third, or those involving the knee-joint, the transfixion must be made through the tibia and not the femur. A short pin, 4 in. long and $\frac{1}{8}$ in. thick, is driven through the crest of the tibia just below the tubercle (*see Fig.* 153, A). Through such a pin very efficient traction can be made, as it pulls upon the actual bony insertion of the extensor and flexor muscle groups of the knee. Another point in favour of the tibial transfixion site is that the pin can be left in place for a long time—six or eight weeks—and for this reason it is well adapted for the treatment of septic open fractures.

FRACTURES OF THE TIBIA AND FIBULA.

Skeletal traction is not required nearly so often in fractures of the leg as in those of the thigh; but nevertheless it is of the utmost value in those cases where it is indicated. Fractures of the tibia and fibula never show the same tendency to great overlapping as do those of the femur. Often the fracture in the early days has only about $\frac{3}{4}$ in. overlap and looks as if simple manipulation would serve to effect reposition; but even under an anæsthetic it often proves to be so difficult that this remains now as the common type of fracture to be subjected to an open plating operation. But it is just in such an obstinate fracture that skeletal traction will prove to be quite a simple remedy.

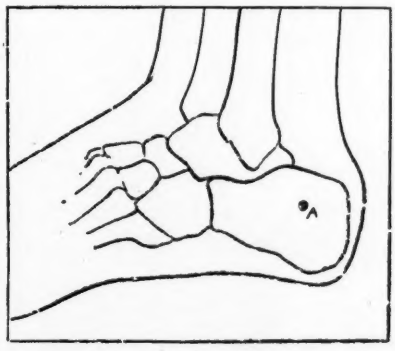


FIG. 156.—Point of transfixion for os calcis. (*From "Modern Methods of Treating Fractures".*)

The transfixion is by the small short pin, which is passed through the posterior process of the calcaneum as far back as possible so as to afford a counter-traction to the pull of the tendo Achillis (*Fig.* 156). The leg must be put up in some form of sling, and I find the short cradle splint (*Fig.* 157) most convenient for this purpose. The weights used are about half those mentioned in the case of the femur—i.e., 10 to 30 lb.—and usually two pulleys are sufficient.

Pin traction through the calcaneum has two special advantages in addition to those of efficiency: the foot is kept in dorsiflexion, because all the pull is made on the heel; and as the traction is made through the ligaments of the ankle-joint, the latter is kept open and supple.

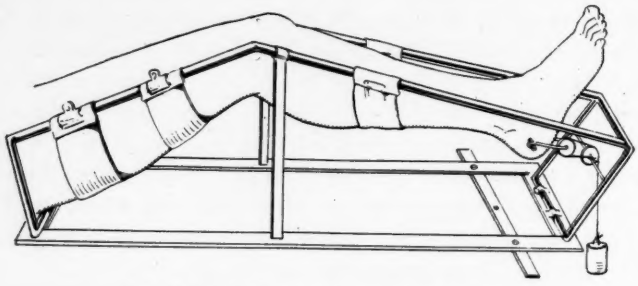
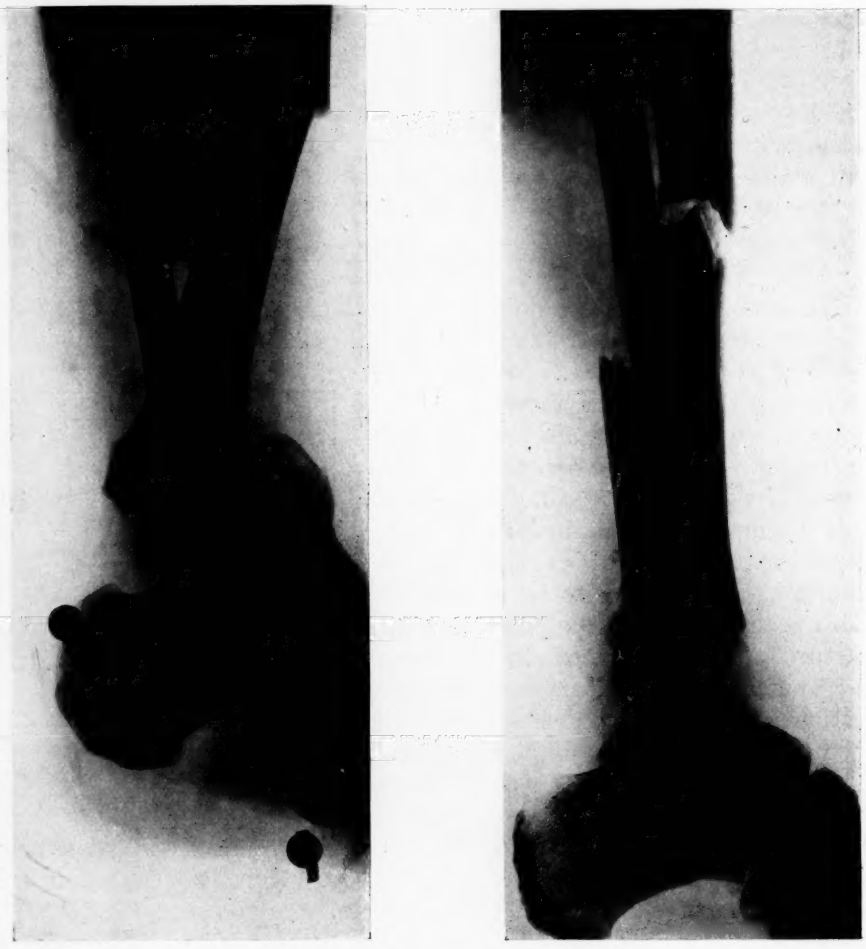


FIG. 157.—Author's short cradle splint.



FIGS. 158 and 159.—Skiagrams of fracture-dislocation of tibia and fibula, before and after treatment.

In two types of case it is better to use a big pin and to transfix the malleoli rather than the heel bone. These are fractures of the upper third of the leg bones, and those fracture-dislocations in which the inferior tibio-fibular joint has been damaged, with marked separation of the two bones (*Figs.* 158, 159).

FRACTURES OF THE LOWER END OF THE HUMERUS.

In the upper limb, skeletal traction is very seldom required, but there is one application which may be of great value. This is for the various types of fracture of the humerus just above or into the elbow-joint, especially when there is great swelling of the elbow which contra-indicates any forced flexion. In such a case the arm is slung to an overhead support with the elbow at a right angle, the olecranon is transfixed with a slender short pin (*Fig.* 160), and a weight of about 10 lb. is attached. In this way the displacement of the fracture can be reduced fully, whilst the limb is kept in the best possible position for the restoration of the circulation and the preservation of the elbow movements.

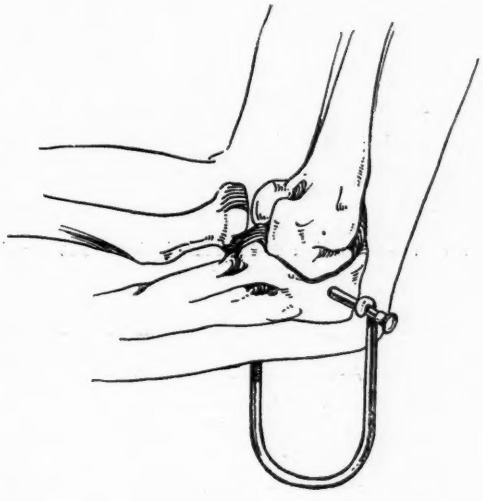


FIG. 160.—Extension of humerus by transfixion of olecranon. (*From "Modern Methods of Treating Fractures".*)

DIRECT SKELETAL ADJUSTMENT OTHER THAN TRACTION.

It not infrequently happens that after skeletal traction has fully reduced or over-reduced overlapping, there still remains some lateral displacement which it is desirable to correct. In the forearm bones lateral displacement is usually the most important element in the deformity, and in the leg bones the displacement of a spiral fracture is notoriously difficult to adjust. This type of lateral displacement can usually be brought into perfect position by means of a simple open exposure, when the bones can be made to interlock. If they will not remain in close contact, then by means of a metal bone bradawl each fragment is partly transfixed and held in position whilst the

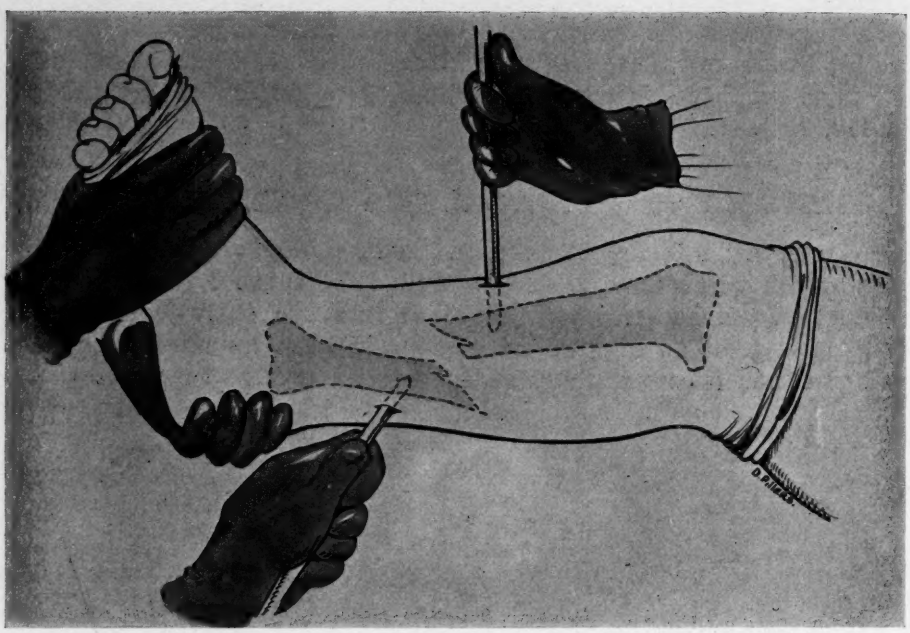


FIG. 161.—Reduction of lateral displacement by the use of bradawls: before reduction.

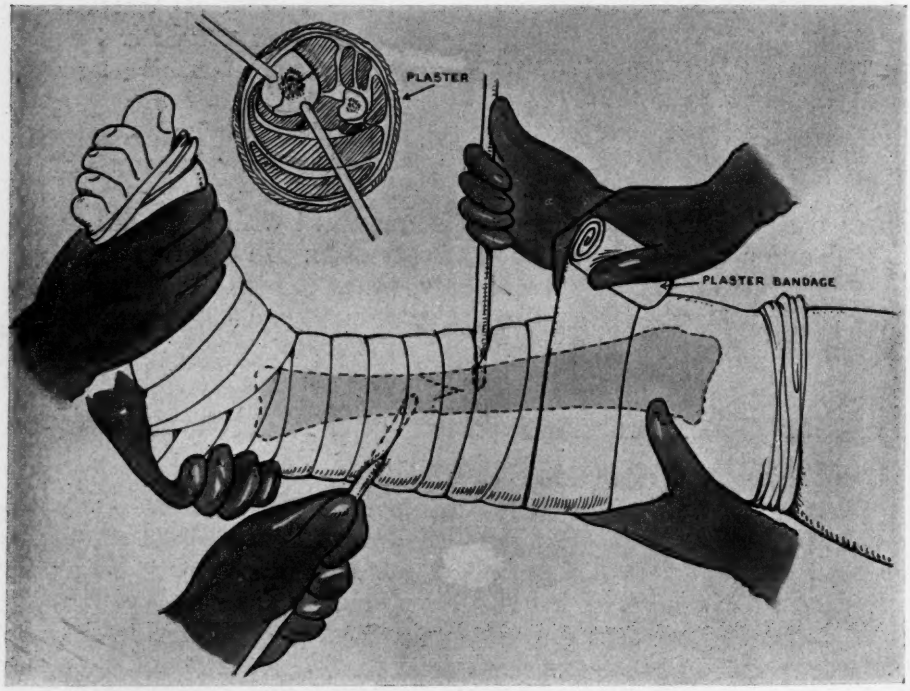


FIG. 162.—The bradawl operation: after reduction and fixation in plaster.

wound is closed and the limb is put in plaster (*Figs.* 161, 162). The plaster surrounds the projecting shafts of the bradawls, which are left in place for

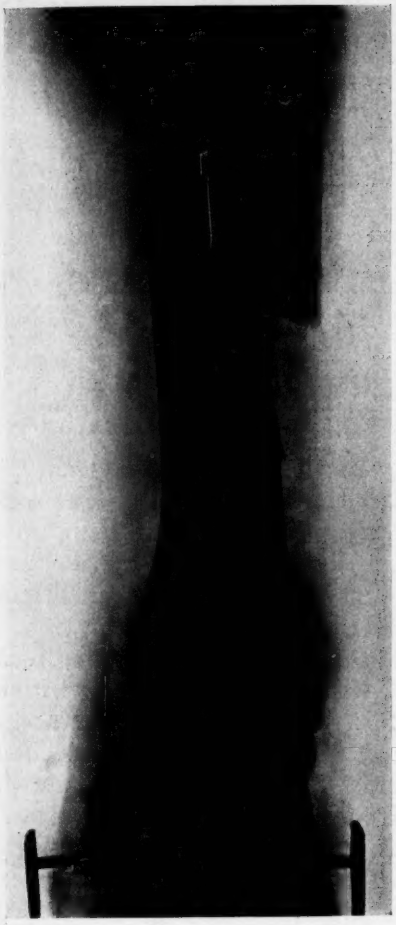


FIG. 163.—Skiagram of fractured leg bones before bradawl operation.

FIG. 164.—The same case four weeks after operation.

twenty-four hours. If this method is used, it is noteworthy how rapid is the natural healing by callus as compared with the delayed union which generally follows the application of a plate and screws (*Figs.* 163, 164).

SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF LARGE SINGLE (NON-PARASITIC) CYST OF THE SPLEEN.

By A. W. SANDERS,
SURGEON TO THE PRETORIA HOSPITAL.

In a paper published in *Surgery, Gynecology and Obstetrics* in November, 1927, by Emil Novak, of Baltimore, it is stated that only seventy-four cases of non-parasitic cyst of the spleen have been recorded. It would therefore seem worth while to place on record another. This case would appear to come under the heading of 'traumatic cysts', judging by its appearance and the pathological report, although there was no history of any injury.

History.—The patient, a girl of 15, who had been perfectly well recently, and who had never had any illness of note, was seized one morning with acute abdominal pain not accurately localized but referred to the lower abdomen. Her temperature rose to 102°, and she looked at first pale and pinched but later on was flushed. The pain was of brief duration and had quite passed off in an hour, the temperature having by that time fallen to normal. No tenderness or rigidity was to be made out, but one found what appeared to be a definite enlargement of the spleen. This organ could be felt extending about an inch and a half below the costal margin, occasioning a slight fullness and prominence in the left hypochondriac region. The swelling was not tender, and, indeed, there was nothing to indicate that it was in any way associated with the severe pain and rise of temperature. There was no history of any injury recent or remote. A cytological examination of the blood showed nothing abnormal.

The patient was kept under observation and examined from time to time, and it was found that the tumour was enlarging slowly, but no symptoms occurred to give rise to any anxiety. An X-ray examination gave no information as to the nature of the tumour.

Some weeks after the discovery of the tumour the patient was examined by Professor Ritchie Thompson, of Johannesburg, who found that it could be displaced forwards and to the right as far as the mid-line. It was then felt to be rounded in shape. No distinct notch could be made out, but it was evidently attached to the splenic region by a definite pedicle. The tumour did not occasion the least pain or discomfort, and the girl looked and felt

perfectly well. Beyond limiting her freedom as to active exercise, particularly gymnastic, no precautions were adopted. The possibility of the tumour being of renal origin was considered. There was an entire absence of renal symptoms, and the mobility and the situation of the tumour immediately below the costal margin and beneath the abdominal wall made its splenic origin seem much more probable. The steadily increasing size of the tumour impelled a decision in favour of operation. Professor Ritchie Thompson kindly assisted.

Operation.—The abdomen having been opened by a vertical incision about 6 in. long, 1 in. to the left of the mid-line, the tumour was readily exposed. The upper part of the spleen appeared normal in shape and consistency, but the lower half was occupied by a cyst about 4 inches in diameter. The cyst was so thin in places that the dark fluid contents showed clearly through it; in other places it was thick, white, and opaque, as though the peritoneal covering had been the seat of an inflammatory change due to friction against the abdominal wall. The cyst extended deeply into the substance of the spleen—in fact well down to the hilum. The fluid contents were evacuated and found to be dark brown in colour, with glistening particles, possibly fat globules, or, as it was thought, cholesterin crystals; but unfortunately no proper examination of the fluid was made. The cyst was for the most part about $\frac{1}{16}$ in. thick, tough and fibrous in character, and of a reticulated appearance in the deeper or imbedded portion. An attempt being made to shell out the more superficial portion showed that the wall was firmly attached to the splenic tissue, and that to remove it in this manner would cause much hæmorrhage and leave a very ragged damaged surface. Being loth to remove the entire spleen if it could be helped, it was decided to attempt the resection of the lower portion containing the cyst. The hilum in the neighbourhood of the cyst was clamped with two artery forceps, and the portion above grasped in the assistant's fingers. The cyst with a wedge of splenic tissue was then excised. The vessels at the hilum that had been clamped were ligatured. The raw surfaces of the spleen were now brought firmly face to face with mattress sutures of coarse catgut, a few strands of the same sized gut being caught under the loops of the stitch so as to serve as a button and prevent the suture cutting through. The approximated edges were then oversewn with a continuous catgut stitch, and the wound was entirely covered with a flap of the great omentum turned up from below and firmly sewn down to the spleen stump.

The amount of blood lost was but slight; the patient stood the operation well, made an uninterrupted recovery, and has had no symptoms since.

Dr. Pijper, who kindly examined the cyst wall, reported that it was composed of fibrous tissue, and had no epithelial or endothelial lining.

It may be questioned whether it was wise to attempt the saving of a portion of the spleen, and it may be argued that splenectomy would have been a safer procedure; but possibly the last word on the functions of this organ has not yet been said, and to retain half a healthy spleen may be an advantage to the patient.

A CASE OF MULTIPLE CHONDROMATA.

By J. W. THOMSON,
HONORARY SURGEON TO THE CLAYTON HOSPITAL, WAKEFIELD.

The patient, I. K., was a man of 23 years. At the age of 2 it was noticed that there were swellings on the fingers of the left hand and wrist. These swellings increased in size and became painful. When he was 13 years old the left index finger was amputated, and a month later, because of pain and deformity, the left arm was removed above the elbow.

Fig. 165.—Left hand, viewed from in front, at the age of 13. Almost all the phalanges, the metacarpals, and the lower ends of the radius and ulna are the seat of tumour formation.

Fig. 166.—Right hand, from behind, at the age of 23.

Recently a complete radiological examination was made, and disclosed multiple tumours in the right hand, chiefly in the ring and little fingers and in the lower end of the ulna; also in the little toe of the right foot. None of these growths cause any pain or inconvenience. Section of one tumour from the finger shows it to be a pure chondroma.

Fig. 167.—Right foot, at the age of 23.

A CASE OF SARCOMA OF THE ANUS.

By R. J. WILLAN, M.V.O.,

HONORARY SURGEON AND LECTURER IN CLINICAL SURGERY TO THE ROYAL VICTORIA INFIRMARY, NEWCASTLE-UPON-TYNE.

William R., age 43, a waggon-man, was admitted on Dec. 8, 1926, to my wards at the Royal Victoria Infirmary, Newcastle-on-Tyne, with a diagnosis of double irreducible femoral herniæ. He complained of pain in the left groin which he had had for the previous three days. He had noticed a right groin swelling fourteen days, and a left groin swelling three days, before admission. There had been no vomiting or nausea; his bowels were perfectly regular. For years he had had eczema of the right leg. For the previous three years he had suffered from 'piles', which had recently become much worse; there had never been any bleeding. He had lost flesh.

On admission he looked anæmic. In each groin there was a rounded swelling the size of a hen's egg; each swelling was hard, smooth-surfaced,

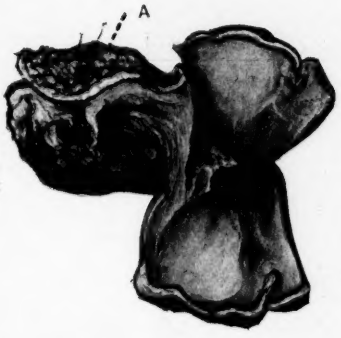


Fig. 168.—Primary sarcomatous hæmorrhoid laid open, with anal skin (A) adjacent.

Fig. 169.—Secondary sarcomatous lymphatic gland of groin laid open.

had a well-defined edge, and was firmly adherent both to the overlying skin and the deeper structures. Neither swelling had any impulse on coughing. He had thrombosed external hæmorrhoids. One of the posterior hæmorrhoids was hard; it was ulcerated on its inner surface, there being growth in excess of destruction in the ulcer. He had dermatitis of the right leg. Nothing abnormal could be found in his abdomen or chest.

During a short period while he was kept under observation both the anal ulceration and the groin swellings visible increased in size. I therefore excised the ulcerating hæmorrhoid and also the huge right groin lymphatic gland (*Figs.* 168, 169). His wound healed, and he left hospital a fortnight later.

Sections were sent to Professor Stuart MacDonald, who reported the tumour to be a small-spindle-celled sarcoma (*Figs.* 170, 171).

After-progress.—His doctor, Dr. Aicken, of Ferryhill, informs me that "the patient, wasted to a skeleton, died from general dissemination of growth

ten weeks after he left hospital. All the time he suffered considerable pain, for which morphia had little or no effect. He also suffered from retention of urine ".

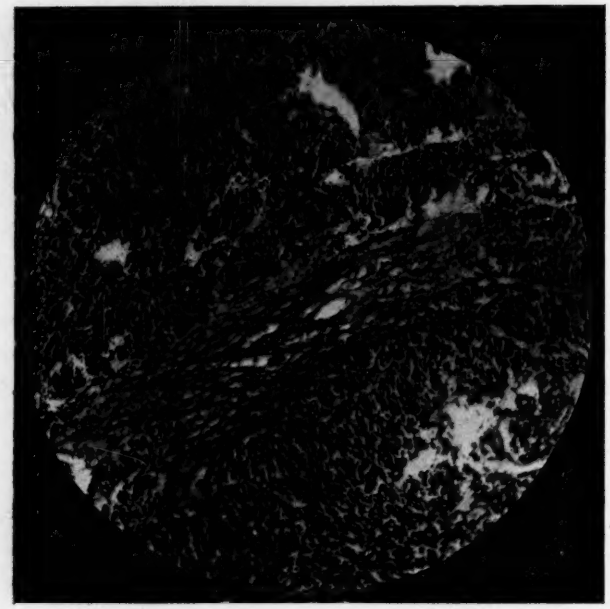


FIG. 170.—Low power view of anal growth. (× 90.)

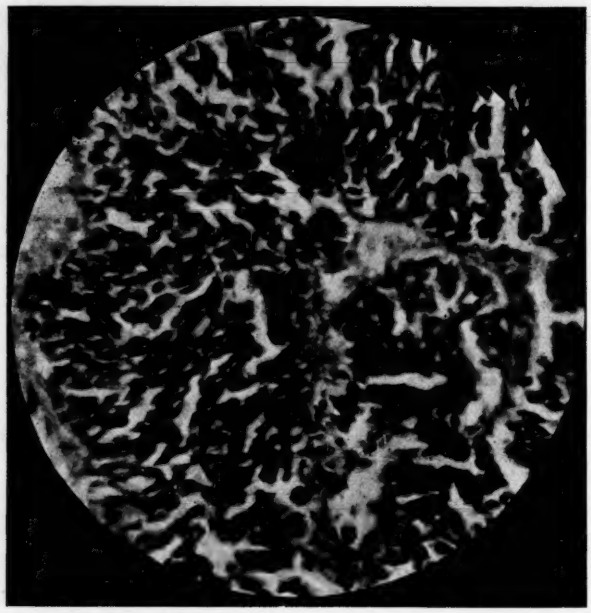


FIG. 171.—Higher power view of anal growth. (× 280.)

I am indebted to Professor Stuart MacDonald for the pathological report and for the microphotographs.

A TUMOUR OF THE CAROTID BODY.

By FRANK HARVEY, London.

Tumours of the carotid body are rare. Clinically, they are recognized as swellings in the neck about the level of the thyroid cartilage, lying either under the sternomastoid muscle or just internal to its anterior border. The swelling is generally regarded as a tuberculous gland, and accurate diagnosis is seldom made; it lies between tuberculous gland, pharyngeal diverticulum, aneurysm of the carotids, adenoma of the thyroid gland, and a branchial cyst. Shipley and Lynn[1] collected and tabulated the reported cases in 1916; and Cahill and Taylor[2] reported a case in 1917. Since then various cases have been put on record, the latest being that of J. E. H. Roberts, reported by M. G. Fitzgerald.[3]

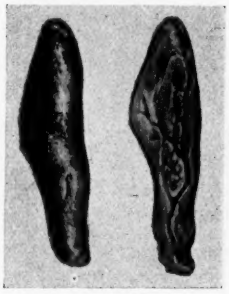


Fig. 172.—Vertical section of collapsed cyst, showing smooth outer wall and irregular inner surface. (*Natural size.*)

Fig. 173.—Section showing patches of pigment on the inner cyst wall. (× 5.)

The following case is that of a lady, age 32 years, whom I saw in consultation with Dr. C. Vere Nicoll, of Frensham, Surrey, on Jan. 16, 1927.

History.—For about a year the patient had noticed a small swelling on the right side of the neck. During the last few weeks and prior to my seeing her the swelling had increased rather rapidly in size, and pain had been experienced, referred to the right ear, shoulder, and clavicular regions. The patient stated that she had never had a severe illness, and had always enjoyed good health.

On Examination.—The patient was a healthy-looking woman. Under the middle of the right sternomastoid muscle could be felt a smooth, tense, non-pulsatile swelling, about the size of a large grape. Mobility was limited. There was no enlargement of the thyroid gland, and the swelling was entirely separate from it. Also there was no movement or increase in size of the swelling when the patient swallowed. No enlarged glands could be palpated on either side of the neck. Auscultation over the swelling was negative.

Operation (Jan. 20, 1927).—A transverse incision was made in the natural crease of the neck at the level of the middle of the anterior border

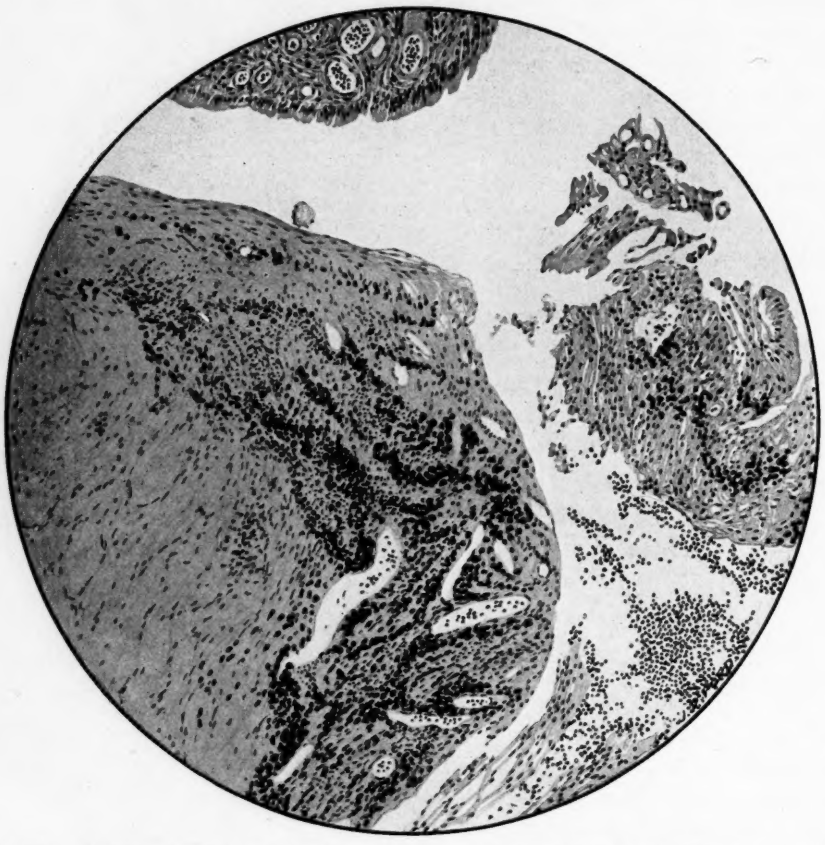


Fig. 174.—Microscopic section showing numerous cells containing blood pigment. (× 65.)

of the right sternomastoid muscle, which was defined and retracted outwards, exposing the tumour. The tumour was dark purple in colour, and was projecting forwards and outwards.

The internal jugular vein was displaced outwards by the swelling. By blunt dissection the tumour was freed from the surrounding structures and was found to arise from the bifurcation of the right common carotid artery. Several moderate-sized vessels from both the external and internal carotid arteries entered the tumour. These were ligatured and the tumour was

removed. The wound was closed without drainage. The patient made an uneventful recovery, and has not since complained of any further discomfort in the neck.

PATHOLOGICAL REPORT.—The specimen (*Fig.* 172) was examined by Dr. A. Osborne, who reports as follows :—

"The cyst is about an inch in diameter. The cavity of the cyst is irregular in shape and the walls are pigmented (*Fig.* 173). Histologically,

FIG. 175.—Microscopic section showing distortion of the polygonal cells, together with fibrosis. (× 65.)

the features are those of a remote hæmorrhage in the carotid body (*Fig.* 174). There is much fibrosis and distortion of the polygonal cells of the carotid gland (*Fig.* 175), and in the fibrous stroma are numerous cells containing blood pigment. The tissue is innocent."

REFERENCES.

[1] SHIPLEY and LYNN, *Jour. Amer. Med. Assoc.*, 1916, lxvi, 1602.
[2] CAHILL and TAYLOR, *Ibid.*, 1917, lxviii, 1898.
[3] FITZGERALD, *St. Bart.'s Hosp. Jour.*, 1927, xxxiv, 62.

THE TREATMENT OF RAYNAUD'S DISEASE BY PERI-ARTERIAL SYMPATHECTOMY.

By HAMILTON BAILEY,

SURGEON TO THE DUDLEY ROAD HOSPITAL, BIRMINGHAM.

For peri-arterial sympathectomy to be efficient from a vascular standpoint, the artery upon which it is performed must be capable of active contraction and dilatation. If the vessel is rigid and calcified, it is unreasonable to expect that the operation will prove of much therapeutic value.

Even in advanced Raynaud's disease the arteries, though distinctly smaller than usual, remain soft and pliable. Here, then, is a condition in which sympathectomy should prove highly effective. A study of two cases has convinced me of the value of peri-arterial sympathectomy in this disease.

Case 1.—An able-bodied seaman, age 44, was invalided from the Royal Navy in April, 1927, with Raynaud's disease. The manifestations were typical in all respects; spasms were occurring frequently, and whilst under observation one could see his tremulous hands go blue; at the same time the fingers down to the interphalangeal joints assumed an ashen hue. The symptoms were largely bilateral, but he complained specially of his right hand, and in September, 1927, the finger-tips of this hand, particularly of the second and third digits, became black, and the skin in this region was obviously commencing to undergo dry gangrene.

On Sept. 15, 1927, peri-arterial sympathectomy was performed on the right brachial artery. The adventitia of the artery was removed for fully three and a quarter inches. At the time of the operation it was noted that the artery was distinctly smaller than normal. There was immediate relief of the symptoms on this side. When seen in November, 1927, the right hand had a normal appearance. The dead, black skin had separated, and a new nail was commencing to grow on the third finger. The patient now complained bitterly of his left hand. On further observation it was interesting to see that the typical spasms were now confined to the unoperated side. On Nov. 19, 1927, peri-arterial sympathectomy was performed on the left brachial artery.

When seen in May, 1928, he stated that he had been completely free from symptoms, and was at work.

This case shows that peri-arterial sympathectomy will quickly relieve the symptoms of Raynaud's disease.

Case 2.—A married woman, age 35, had suffered from syncope of her hands and feet for ten years. Since childhood she had noticed that her hands became blue on the least exposure to cold, but for ten years the typical spasms of Raynaud's disease had occurred spontaneously. In spite of courses of drugs, massage, endocrine therapy, and artificial sunlight, the disease had progressed. The artificial sunlight had intensified a tendency to scleroderma, and general desquamation had occurred for two months after this treatment had ceased.

She came under my notice in January, 1927. The fingers of both hands as far as the nail-beds were shrivelled and black. Many of the toes were similarly affected. Whilst under observation typical spasms of Raynaud's disease were seen on many occasions. As gangrene had progressed further in the right hand than the left, and as she complained more about the right hand than any other limb, peri-arterial sympathectomy was performed on the right brachial artery on Jan. 24, 1927. The artery was very small, about one-sixth the size of a normal vessel. Four days after the operation

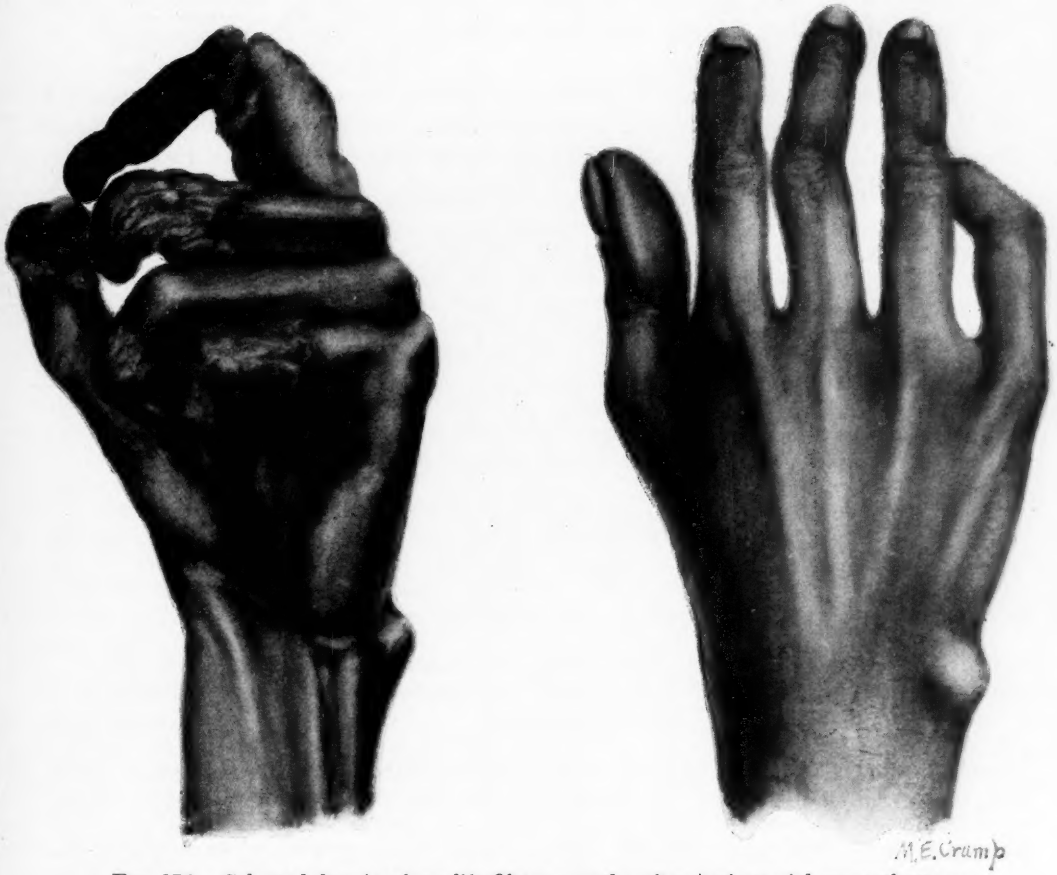


FIG. 176.—Coloured drawing from life fifteen months after peri-arterial sympathectomy on the right brachial artery. The disease has advanced apace on the unoperated side (left), whilst on the side upon which the sympathectomy was performed (right) not only has the disease been arrested, but also there is very marked nutritional improvement.

it was noted that during a paroxysm of the disease the left hand was alone involved.

Six weeks later moist gangrene commenced in the right foot and began to spread rapidly towards the middle of the calf. This was treated by amputation in the lower third of the thigh, preceded by a sympathectomy on the femoral artery. She made a slow recovery and refused any further operative treatment.

I have kept the patient under intermittent observation. The disease has progressed very extensively in the left hand and the left foot, whereas the right hand, which originally presented the most advanced signs and of which the patient most complained, now presents a comparatively normal appearance and is the only useful member. The illustration (*Fig.* 176) was drawn from life fifteen months after peri-arterial sympathectomy had been performed on the right brachial artery.

This case is unique, for we have in the unoperated side a control. Upon the operated side we find that the disease has not only been arrested, but also, after an interval of fifteen months, shows very considerable improvement, whilst on the unoperated side it has advanced apace.

The conclusions are: (1) That peri-arterial sympathectomy performed on the main artery supplying a limb will quickly alleviate the symptoms of Raynaud's disease in that limb; (2) That there is a certain amount of evidence to justify the hope that this relief is permanent.

REVIEWS AND NOTICES OF BOOKS.

Clinical Researches in Acute Abdominal Disease. By ZACHARY COPE, B.A., M.D., M.S. (Lond.), F.R.C.S., Senior Surgeon to Out-patients, St. Mary's Hospital, London. Second edition. Pp. 214 + xvi, with 44 illustrations. 1927. London: Humphrey Milford. 10s. 6d. net.

THE second edition of this book contains three new chapters—one on extravasation of bile, one on acute ascending parenchymatous enteritis causing paralytic ileus, and one on shock and collapse, with special reference to acute abdominal disease.

In the chapter on extravasation of bile, cases that have come under the personal care of the author are quoted. The condition is divided into retroperitoneal and intraperitoneal extravasations, the latter of which are the commoner. The different views as to the state of affairs that allows of the extravasation are shortly described —that of filtration and that of the presence of a minute undiscovered perforation of the bile-ducts or gall-bladder. On the whole the latter is probably the cause in most cases, although in many of those mentioned no perforation either at operation or at autopsy could be discovered. The clinical symptoms are variable, but one especially mentioned by the author is the remission of symptoms after the first acute onset. This may be due to the temporary stoppage of leakage, by the omentum, of bile which is secreted under very low pressure. At present our knowledge of the early signs and symptoms of this condition is not very accurate, and this is borne out by the fact that the mortality in the author's cases amounted to no less than 60 per cent.

The longest chapter in the book is a new one on "Shock and Collapse". This was given by the author as a Hunterian Lecture at the Royal College of Surgeons. He points out the still existing difference of opinion both as to the definition and essential nature of the condition of 'shock'. The great difficulty seems to be the absence of a suitable definition of the word 'shock', especially from the purely clinical point of view.

The clinical symptoms of shock are grouped into three classes: (1) Those due to circulatory failure: pallor, superficial cold, mental dullness, increased pulse-rate, diminished pulse-volume, lowered blood-pressure. (2) Those due to depressed metabolism: subnormal temperature, diminished secretions and excretions. (3) Those due to sympathetic stimulation: sweating with consequent thirst, dilated pupils. The author says that these symptoms may be dissociated in varying degree, but in demonstrable shock there is nearly always either subnormal temperature or a lowering of blood-pressure, and usually both. To this we would add an increased pulse-rate, as he does in his first group of clinical symptoms.

Several authors and cases are quoted to prove that a patient with a slow pulse may be suffering from shock, but these cases seem to be exceptional. In the same way authors are quoted to prove that a low blood-pressure is not an essential accompaniment of the clinical picture of shock. For instance, "It is probable that shock may be present before the blood-pressure falls, though it is difficult to recognize its presence except that the patient may look bad". Again, "Although most shocked patients have a low blood-pressure and rapid pulse, there are some notable exceptions". This latter the author considers to be the stage of latent shock.

Perforations of the duodenum and stomach are specially mentioned as causing the *appearance* of shock, and Sir Berkeley Moynihan is quoted to substantiate this statement, though this authority denies the existence of 'shock' in the true surgical sense. The author agrees that difference of opinion depends to a certain extent on the definition of the word 'shock'. Either that definition is imperfect or the

proof of its correctness should be specially demonstrated by careful and detailed data. We are inclined to think that the former is the cause of this difference of opinion. We agree with Sir Berkeley Moynihan that in the early stages of perforation of duodenal ulcer the absence of a rapid pulse, lowered blood-pressure, and diminished blood-volume in the vast majority of cases should preclude the use of the word 'shock', a word which to the majority of practitioners calls up a picture of a rapid pulse and a lowered blood-pressure. Cases are mentioned as suffering from what the author calls ' shock ' who have a slow pulse and a normal blood-pressure, but we think these are exceptions to the general rule. He says that all authorities agree that shock is present in cases of acute pancreatitis—that is, the pulse is rapid from the onset of the symptoms—but quotes one case in which the pulse was slow and which was operated upon as a likely case of perforation of a duodenal ulcer. This again was an exception to the general rule that shock is usually accompanied by a rapid pulse. Patients suffering from a perforation of a duodenal ulcer do not in the early stages show the essential signs of what we consider to be usually accepted as those of shock—that is, a rapid pulse, a lowered blood-pressure, and a diminished blood-volume.

The picture of a patient suffering from shock with a rapid pulse and a lowered blood-pressure is so firmly impressed upon the mind of the average practitioner, that we think it would be wiser to use some other expression to denote the condition of a patient in the early stages of perforation of a duodenal ulcer. Such an expression as 'prostration' might be adopted, as was suggested by Sir Berkeley Moynihan in his latest article in the *Practitioner*, January, 1928, on perforations of gastric and duodenal ulcers. He there says shock in the surgical sense is absent. The word 'shock', used in its strict sense, means a condition in which increased pulse-rate, diminished blood-pressure, and reduced blood-volume are present. None of these is found in the first hour or two after a perforation has occurred. Shock comes on later—when peritonitis has developed. This, we think, represents the true state of affairs in the very large majority of cases, and we would suggest that perhaps the word 'prostration' more clearly defines the condition of a patient in the early stages of a perforation.

The book is one which we can strongly recommend to those who are interested in the more scientific side of that very important subject, the acute abdomen.

Die Leitungsbahnen des Schmerzgefühls und die chirurgische Behandlung der Schmerzzustände. By Professor Dr. O. FOERSTER, Breslau. Royal 8vo. Pp. 360, with 104 illustrations. 1927. Berlin and Vienna: Urban and Schwartzenberg. Sewed, M. 19.50; bound, M. 21.90.

A BOOK on pain from the pen of Professor O. Foerster is naturally an event in current medical literature, and his new book *The Pathways of Pain and the Surgical Treatment of the Algesias* is in many respects the most important work on the subject which has yet appeared.

It is divided into two sections, in the first of which the anatomical paths of pain, from the receptors through the peripheral nerves into the posterior roots and posterior horns, are followed step by step in considerable detail, and the applied physiology of the transmission of pain is discussed. In six further chapters the pathways are traced through the cord and brain-stem to the final end-stations in the thalamus and cortex cerebri. In the second division of the book pain is discussed in its clinical aspects in so far as it is occasioned by affections of the different segments of the conducting mechanism.

Foerster's name has latterly become associated with the idea that the vessels are media for pain conduction. Peri-arterial sympathectomy has taught us that the great vessels are sensitive in a high degree, though apparently not all to an equal extent. Foerster mentions that L. R. Müller denies pain fibres to the cerebral vessels, but Foerster has found ligature of these vessels frequently productive of pain, as for instance during local excision of cortex. He has also found the injection of an

angiodilator (papaverine), or direct injection of calcium bromide into the carotid, an instantaneous cure for migraine. He mentions a most interesting case of rupture of the lower roots of the brachial plexus (C 7, T 1) distal to the entrance of the rami communicantes. Although the skin was anæsthetic from the third to the fifth finger, deep pain was not abolished. He exposed a digital nerve and stimulated it with the faradic current without producing any pain. He then freed the corresponding digital artery, and electrical stimulation of that immediately produced severe pain. Foerster restates his views on the sensibility of the dura in view of Müller's criticism, and asserts that the dura of the cranial base and of the tentorium is exquisitely sensitive to cutting and sometimes even to stroking, but that it becomes less and less sensitive towards the vertex. The dura in the region of the main trunks of the middle meningeal arteries is also very tender. Experience varies on these points, and indeed the author himself admits that there is no constancy in these findings from one case to the next. In contrast to this he has not been able to produce pain from stimulation of the pia mater even when it was inflamed. The spinal theca he finds less sensitive to pain than the dura mater.

When we come to the posterior roots, we find a number of most helpful photographs of patients with the areas of sensory defect after root-section most carefully plotted out, and these in themselves are a most valuable contribution. Some of the delimitations have been obtained by the reverse method—electrical stimulation of the cut peripheral end of a root, for this is followed by an immediate outspoken vasodilatation of the skin, thus enabling one to identify accurately the particular root cut and to satisfy oneself that a sufficiently wide area is being denervated.

On the vexed question as to whether or not the anterior roots conduct sensation or any part of sensation, Foerster makes some interesting observations. He regards the anterior roots as an auxiliary sensory pathway ('akcessorische Hilfsbahn'). In a diagram illustrating the afferent fibres of the anterior root he draws a branch of a posterior root axon and a sympathetic fibre entering the radix anterior. He has produced severe pain following stimulation of the central end of a cut anterior root. It varies from one nerve to another. In one case he cut the dorsal roots 6 to 10, and whilst the 8th anterior root on stimulation produced maddening pain, in a parallel case a scarcely painful unpleasant feeling resulted from a similar stimulation. In the first case, after the cutting of the posterior roots, deep sensibility had been very little altered, in the second profoundly changed. It is well known that division of the sensory root of the Gasserian ganglion does not abolish deep sensibility in the face, but Foerster has found that by dividing the hypoglossal as well, pressure pain was abolished in half the tongue. He has also found the central end of the facial nerve to produce lively pain on stimulation. As for the existence of an extra-radicular pain pathway in the sympathetic, he mentions a case in which he extirpated the Gasserian ganglion, cut the cervical sympathetic, and performed a peri-arterial sympathectomy on all the carotid arteries. The result was that pressure sense and pressure pain were diminished but not abolished. Foerster goes on to discuss the pathway of deep sensibility in the treatment of gastric crisis. It is interesting to find him producing clinical evidence of pain from lesions of the posterior column, a fact with which all will be familiar who have read the account which Brown-Séquard gave of his own famous animal experiments.

A most excellent section is the one on posterior horn pain, containing several observations on pain arising from central gliomatosis. He comments on the frequency of spontaneous pain in intramedullary tumours, and says that in all the cases of this kind which he has observed, pain in the dermatomes corresponding to the site of the tumour was the first symptom. He refers particularly to a cylindrical central glioma which began in the middle of the thoracic cord and spread upwards and downwards from there. Shortly before death, analgesia and thermanæsthesia extended on both sides from the 2nd cervical to the 4th lumbar segments. Its gradual spread upwards and downwards was traceable clinically by the development of spontaneous pain in successive dermatomes, to be followed by analgesia. He has observed the same march in syringobulbia, and has watched pain spread from the trigeminal field to C 2–4.

He records observations confirming the fact that pain and temperature sense may be dissociated in the posterior horns no less than in the spinothalamic tract, and the temperature sense itself may be dissociated into hot and cold elements of different distributions. We can allow ourselves only one more quotation. He now states categorically that the pain fibres in the crossed spinothalamic tract need only one segment to reach their position, and not the three or four which it is the common usage to allow them. With this statement the reviewer finds himself in complete agreement. Foerster mentions, for example, a case of bilateral section of this tract at the upper end of the 5th thoracic segment, and all dermatomes from T 6 to S 3 were analgesic and thermanæsthetic. There can be no doubt in our mind that the depth of the cut is the most important factor in determining the level of sensory defect, and Foerster appears to be correct in assigning the most superficial position to the fibres from the lowest levels, whilst those from higher levels are much deeper. In his schema of the sensory and motor pathways in cross-section he shows lamination of the main sensory pathways (which we can accept in great part), but he also laminates the pyramidal tract in the cord, and for this we can find no justification.

Foerster appears to have antedated Spiller and Frazier in section of the spinothalamic tract, for he mentions performing this operation in 1912 for tabetic pain. He states (pp. 108 and 109) that the first experimental knowledge on the spinothalamic tract as a pathway for pain was obtained by himself by accident. In injecting a case of intercostal neuralgia in the 6th intervertebral foramen the needle point went in too deep and entered the cord, the patient complaining of terrible pain in the opposite leg, and he mentions that severe pain may follow any handling of this tract.

So much for the general arrangement; it is clear that we have here a fuller account of the pain-conducting system than exists in any monograph heretofore. It is a book which everyone interested in pain and its treatment should have and reflect on. We have quoted freely from the monograph, since there is so much clinical fascination to be found in it, for the general surgeon no less than for the neurologist.

It is clear that Professor Foerster has allowed no opportunity to pass for enriching his own experience, and we are grateful to him for the knowledge which he has passed on. It seems ungracious to complain, but why is it that even the most important of Continental books are allowed to appear without an index? The volume is illustrated by a great number of photographs and diagrams, all of which have been most painstakingly prepared.

"L'Anæsthésie Régionale." By VICTOR PAUCHET, P. SOURDAT, G. LABAT, and R. DE BUTLER D'ORMONT. Fourth edition. Royal 8vo. Pp. 296, with 328 illustrations. 1927. Paris: Gaston Doin et Cie. Fr. 55.

MANY books have appeared in recent years dealing with the subject of regional anæsthesia, but this one, which has now reached a fourth edition, still retains its place in the forefront. To read any book on this subject is to realize how easy it is to produce a perfect anæsthesia; it is not until one tries it that the difficulties and disappointments present themselves. Dr. Pauchet sums up the position very well when he says: "Do not condemn it untried. Its users are constantly making advances: actually there is not one student of medicine who can ignore this method of anæsthesia. There is not a surgeon who can do without it in his practice. Those who condemn it, do not know it. To know it, one must learn, and this book will teach the methods."

This last claim is well founded. No trouble is spared in the text to make every step as clear as possible, and every procedure is amply illustrated by the very numerous plates. The method of arrangement is good: an account is given in the beginning of the method of anæsthetizing any nerve-trunk in the body, and in the later part of the book the operations which can be performed on the different parts of the body are considered in turn. In each case the nerves which must be dealt with are enumerated, and one is referred back to the earlier pages for details of the

technique. Unfortunately in several places the cross-references are omitted, an error in the editing which, although trivial, is rather irritating.

The book has been well produced, in particular the illustrations, which are both numerous and instructive. As a text-book for the beginner, or as a book of reference for the more expert, we can confidently recommend this work.

Local Anæsthesia. By G. DE TAKATS, M.D., M.S., Assistant Professor of Surgery, Northwestern University Medical School, Chicago. With a Foreword by ALLEN B. KANAVEL, A.B., M.D., D.Sc., Professor of Surgery, North-western University Medical School. Medium 8vo. Pp. 221, illustrated. 1928. London: W. B. Saunders Co. 18s. net.

THE use of local anæsthesia would seem to be extending in America. Certainly local methods often present advantages. The author of this book graduated in local anæsthesia in Budapest and has continued his experience amongst the American people; he can therefore speak with some authority. His book is a summary of present-day practice. It is not too long, and is very clear in its directions. He is dogmatic in his statements, but they are always carefully considered. We welcome particularly a book on this subject on which the author is not over-enthusiastic. De Takats does not recommend paravertebral or posterior splanchnic anæsthesia for abdominal work, believing them to be unsafe. He has also seen accidents from deep cervical block anæsthesia. For thyroidectomy he substitutes a combination of superficial cervical block with injection round the upper poles of the gland. Though individual surgeons will not agree with all the statements made in the book, it can be recommended as a concise reliable guide to those inexperienced in this method of anæsthesia. In the last line of page 174 'cervical' should read 'dorsal'.

Bone Sarcoma. By H. KOLODNY, Ph.D., M.D., Department of Surgery, State University of Iowa. Royal 8vo. Pp. 214, with 104 illustrations and 41 plates. 1927. Chicago: Surgical Publishing Company.

THIS monograph is based almost entirely upon the material from the Registry of Bone Sarcoma of the American College of Surgeons. In this Registry, which was started in 1920, no less than 700 cases have been tabulated. The author gives reasons for rejecting the parasitic origin of sarcoma, and for considering it to be due to an alteration in growth restraint after trauma.

It was inevitable that any new review of a complex subject such as this should lead to a proposal for fresh classification, and equally inevitable that much of such new classification would prove very unacceptable to other workers. In the first place, all the ordinary malignant sarcomas the tissue of which arises from bone-forming elements are termed osteogenic sarcoma. Thus the time-honoured distinction of spindle-cell or round-cell, as well as that of periosteal or endosteal, are abolished, and for this we think that full and convincing reasons are given. It is the same as the abolition of the distinction between periostitis and osteomyelitis in acute affections of the bone. Periosteal fibrosarcoma is admitted to be a real entity, and is a tumour of low malignancy of extraperiosteal origin. The greatest difficulty to the English reader is presented by what is termed Ewing's tumour or Ewing's sarcoma. This is described as a malignant endothelial growth occurring in the mid-shaft of the pipe bones and in the small bones. It arises in young patients and is usually multiple. It closely resembles chronic osteomyelitis in its X-ray appearance. In addition to the usual clinical evidences of pain following trauma, the cases present repeated febrile attacks, and if it were not for the absence of leucocytosis the condition might easily be confounded with leukæmia. If time should confirm the reality of this as a separate entity it is most sincerely to be hoped that a suitable descriptive name may be found for it. Myeloma is a name reserved for cases of multiple bone-marrow tumours associated with Bence-Jones albumosuria, and the tumour which English writers usually call myeloma is called a giant-cell tumour.

The largest section of the book is devoted to the description of osteogenic

sarcoma, and 41 pages are given to an account of the multiform histology of this disease. The microphotographs, and indeed all the illustrations, are models of clearness and of excellent reproduction. Two little points of criticism we would suggest, however—that the figures should be numbered consecutively throughout, instead of being separately numbered as plates or figures, and also that the degree of magnification of the microphotographs should be indicated. Much statistical and clinical information of great interest is given. It is reckoned that about 1 in every 100,000 of the population is affected with bone sarcoma. Males to females are as 4 to 3. The age of maximum frequency in both sexes is 20. The proportion of cases in the arm and leg is as 2 to 11; the right side to the left side as 5 to 4. The order of frequency in individual bones is as follows: femur, tibia, humerus, pelvis, fibula, shoulder girdle, ulna, hands, feet, ribs, skull, jaws, and vertebræ. There has been no case registered in the radius or lower end of the tibia. About 5 per cent of all bone sarcomata occur in Paget's disease. Pain precedes tumour formation by an interval of days, weeks, or months; it is worse at night and is relieved by radiation. Trauma, which is usually a trivial sprain or contusion, precedes symptoms by about one month. Radiation has such a definite effect in allaying pain and in making the X-ray picture more sharply defined that it is regarded as an important diagnostic test. Biopsy should never be employed as a diagnostic test except under the precaution of a double elastic tourniquet, and then only as a preliminary to amputation if the section should reveal a malignant growth. The author suggests that the best routine treatment should be: (1) Radiation followed by splinting and recumbency; (2) Amputation; and (3) Prolonged prophylactic radiation of the chest. He considers that the cellular structure is of very little value in prognosis.

The last 45 pages are devoted to an account of the giant-celled tumour, which is of benign nature and which can usually be treated by conservative methods.

The wealth of illustrations and of clinical information make this monograph one of great value for reading and for reference.

Neoplastic Diseases: A Treatise on Tumours. By JAMES EWING, A.M., M.D., Sc.D., Professor of Pathology at Cornell University Medical College, N.Y.; Pathologist to the Memorial Hospital. Third edition. Royal 8vo. Pp. 1127, with 546 illustrations. 1928. Philadelphia and London: W. B. Saunders Co. Ltd. 63s. net.

THIS is the third edition of a book which requires no introduction; for the student, and more particularly the practising pathologist, already knows it as a valuable book of reference, and an amazingly complete guide to the literature of tumours.

When comparing this with the preceding editions it is plain that the book has been very carefully revised; and there have been so many changes, great and small, in the framing of individual paragraphs, that one might almost say that the book has been re-written. The illustrations, which are excellent, have also been gone over, and many new ones have been added.

Innocent as well as malignant tumours are described, though the object of the work still stands out—to be a study and record of cancer. The scope of the work is widened still further by the inclusion of certain conditions (especially in the section on bones) which lie in the borderland between inflammation and true tumour formation, and diseases of the endocrine organs which are more functional errors than neoplasms.

The sections dealing with the theories of the nature of cancer, experimental cancer research, sarcomas and endotheliomas of bone, and brain tumours, have been almost completely re-written and brought up to date. Many new facts and illustrations appear in the descriptions of neurofibromatosis, liposarcoma, chordoma, carcinoma of the thyroid, and pineal gland tumours.

Although the subject is dealt with primarily with a view to expounding the natural history and the structure of neoplasms, yet frequent reference is made to the clinical aspects of tumour formation, and this is of the utmost value to the clinician as well as to the pathologist.

The main criticism we would offer is in regard to nomenclature. When reading certain chapters—for instance, those on the tumours of bone and on renal tumours

—it is not possible for a person brought up on English books to follow Dr. Ewing's descriptions until he has 'translated' the terms into another language. This problem of nomenclature in pathology is one which demands the serious consideration of investigators and teachers, so that the science of pathology may be international, and that new work may be appreciated by workers in the same field all over the world.

Allgemeine und specielle chirurgische Operationslehre. By Professor MARTIN KIRSCHNER and Professor ALFRED SCHUBERT, Königsberg. Royal 8vo. Vol. I: General Surgery. Pp. 648, with 709 illustrations, for the most part in colour. 1927. Berlin: Julius Springer. M. 114; bound, M. 120.

THERE can be no doubt as to the importance of this new German work. At the first glance one realizes that it represents an effort to attain the very best that is possible in text and figures, and further detailed perusal serves to confirm this first impression. Although large and comprehensive, the book is the product of only two authors, and in this respect it compares very favourably with those systematic treatises which emanate from a great number of specialists. There is a uniformity and proportion in the various chapters which is lacking in the composite text-book.

The first chapters of over one hundred pages are concerned with general principles of the operation room, the investigation of the patient before operation, and the methods of placing the patient and of wound suture.

The second section is devoted to the abolition of pain by the various methods of anæsthesia. In regard to the descriptions of inhalation anæsthesia, we would remark that the usual intratracheal methods are not given due prominence, although full mention is made of the use of Kuhn's and of Hahn's laryngeal tubes. All methods of local anæsthesia, whether by infiltration or by nerve blocking, are fully described, and the beauty and accuracy of the drawings make this section very valuable. Then follow chapters on aseptic technique and the operative treatment of local infection; the arrest of hæmorrhage by forceps, ligature, and tourniquet, and the treatment of the effects of loss of blood by saline and blood transfusion. The fifth chapter, on operations on the skin and subcutaneous tissues, is introduced by some most useful and beautifully drawn figures of the lines of tissue tension in the skin over all parts of the body. There follows an excellent and fully illustrated description of various plastic operations, including the use of reversible tube-flaps and methods of epithelializing cavities and sinuses. The sixth chapter gives a complete account of operations for tendon transplantation, including the repair of tendons by free fascia flaps. The eighth chapter, on affections of the blood-vessels, mentions a method of treating cavernous angiomata by implantation of bits of metallic magnesium which is new to us. Operative proceedings on varicose veins are given in great detail; injection methods are commended, but only in conjunction with ligature of the internal saphenous vein; no fewer than seven other operations are described and illustrated, and this is an example of the value of this work for its completeness as a book of reference. The next chapter is on the surgery of the peripheral nerves, and is very complete. It includes all the classical procedures for nerve-bridging, together with several new suggestions, e.g., that for dealing with an amputation neuroma by implanting the freshened nerve-end into the side of the nerve-trunk. The figure (520) of peri-arterial sympathectomy is a good example of the combination of anatomical accuracy and artistic merit which is a great feature of this work. The presentation of essential parts in colour whilst the main drawing is in black and white gives a very good effect. The last three chapters deal with operations on bones, joints, and amputations. They are admirable in the clearness of description and in the wealth of illustration. Elaborate methods of bone suture by wire receive, we think, attention which they do not merit. Methods of comminution osteotomy are described which are unusual, but their value is obvious. In describing arthroplasty general principles are given in connection with the knee and elbow, but we should like to have had full details about the hip-joint.

The excellence of this first part of the work makes us eager to receive the second volume dealing with operations upon special organs.

Emergency Surgery. By GEORGE DE TARNOWSKY, M.D., F.A.C.S., D.S.M., Col. M.C., Professor of Clinical Surgery in the Loyola University Medical School. Medium 8vo. Pp. 718 + xvi, with 324 illustrations. 1926. Philadelphia and New York: Lea & Febiger. $7.50.

THE title of this book may prove a trifle misleading to those who look to find an account of the general emergencies in surgery. It deals only with injuries and wounds, the result of accidents. The book is intended primarily for the instruction of industrial surgeons, and is written by one who has had an extensive experience in all sorts of injuries, including battle casualties and the accidents of civil life.

A great deal of interest is now being taken in industrial medicine, and employers of labour and big corporations are finding that "the medical care and attention given to an injured worker determine to a large degree the time of his return to productive employment". So gradually institutions for the care of workers, and literature on the subject, are beginning to spring up, and Mr. Tarnowsky has produced a long, detailed account of what to do in every kind and grade of wound from the crown of the head to the sole of the foot. One cannot help being reminded of the type of book known in the past as "Wund Artznei" in which the mediæval students were instructed how to deal with injuries of the head or stab wounds, and every variety of contusion and dislocation. This book is in fact an up-to-date complete instructor in all that pertains to wounds and accidents, and it may prove of inestimable value to many who are called upon to treat accidents. There is one chapter whose importance is rather difficult to assess, namely, that dealing with the medico-legal aspects of surgery; though the laws of the United States and Great Britain are not identical, still this chapter seems worth reading.

The Tongue and its Diseases. By DUNCAN C. L. FITZWILLIAMS, C.M.G., M.D., Ch.M., F.R.C.S., Surgeon and Lecturer on Surgery to St. Mary's Hospital. Demy 8vo. Pp. 505, illustrated. 1927. London: Humphrey Milford. 36s. net.

THIS volume sets forth what the author believes to be the modern surgery of the tongue, based upon a wealth of clinical and pathological material and an astounding amount of information culled from English and foreign literature. Every section of the book contains detailed references to and abstracts from the published accounts of diseases of the tongue, and a very full bibliography is appended to every chapter. We must confess that although these references are of interest, yet the greater number of them cannot be of much value except to those who, when meeting with a rarity, may be anxious to discover where and when it has been met before. All such must be deeply indebted to Mr. Fitzwilliams for his wide review of the literature.

The early chapters deal with the anatomy and development of the tongue, wounds, and acute and chronic inflammation. Then follow four chapters on the salivary glands, ranula, dermoids, and thyroglossal tumours and cysts, which are, in our opinion, the best in the book. They are well illustrated, and deal with these somewhat confusing subjects in a clear and practical manner.

The rest of the work is devoted almost entirely to cancer of the tongue. The author states in the preface that "the chief lesson of this book is that cancer of the tongue . . . is just as curable in its early stages as is cancer elsewhere". Although it is stated (no doubt correctly) that in early diagnosis and treatment lies the only hope for better results in the surgery of cancer of the tongue, yet we cannot find any evidence produced by the author from which we may learn the chief lesson of the book. The description of operations is followed by figures from many sources, which show merely that a large percentage of patients present themselves for treatment when their disease has become inoperable; and that of those operated upon, only a small percentage survive for three years.

In dealing with the two-stage operation, the advantages of carrying out the removal of the tongue first and the glands later are dwelt upon, without reference to the drawbacks of this procedure. Diathermy is regarded with disfavour, and it appears to us that insufficient stress has been laid upon treatment by radium, using modern technique. No mention is made of the adjuvants to anæsthesia which tend to reduce the number of inoperable cases.

By its nature and its cost the book is rather a work of reference for the specialist than a text-book for the student. Under the circumstances the illustrations should be improved to conform to the standard of the rest of the work.

Curieterapia: Basi Fisiche e Biologiche: Applicazioni. By CARLO PEDRAZZI. Demy 8vo. Pp. 130, illustrated. 1927. Bologna: Licinio Cappelli. L. 35.

THIS monograph deals with the physics, the biological effects, and the applications of irradiation. It is based mainly upon the technique and general work of the Paris Radium Institute, and gives a readable and reliable account of radio-activity, radio-sensibility, and the principles of treatment. Rather more than half the volume is devoted to accounts of regional treatment as practised by Regaud and already well known. Even the illustrative photographs are mainly borrowed from French sources. There is a bibliography of ten pages.

Le Fratture del Gomito. By F. ROSSI. With a Preface by Professor BALDO ROSSI. Medium 8vo. Pp. 312, with 246 illustrations. 1927. Milan: Coop. Grafica degli Operai.

THIS monograph, based on 503 cases of fractures about the elbow-joint, has as its object to furnish evidence from Professor Baldo Rossi's clinic at the Ospedale Maggiore that results as good as any to be attained by operative methods may be achieved without the knife, by careful radiography, skilful reduction, and unremitting attention to after-treatment.

A great many cases of supracondylar fracture are illustrated by serial radiographs, and well sustain the claims made for non-operative measures, whether treated by the acute-angle method or by weight extension, as conditions necessitate. The clinic is not without its difficulties and problems, but the number of cases of ankylosis, false joint, and deformity appears to be small. In fractures of the external condyle the author states that it is only rarely necessary to resort to operation if repeated manipulations with X-ray control be perseveringly adopted.

Of eleven cases of fracture of the internal epicondyle, nine were treated without operation and with entire success; and much the same may be said of the cases of fracture of the whole internal condyle, though the difficulty of preventing ultimate deformity is recognized. T-shaped fractures can generally be manipulated into place and maintained by a posterior plaster splint, but extension may be necessary. Separation, partial or complete, of the epiphyses, is treated by immobilization for as short a time as possible, followed by the most careful passive movements.

The latter part of the book deals with fractures of the upper ends of the radius and ulna; in every case details of the manipulative reduction, duration of treatment, and end-results are given, and cases illustrated.

As a work of reference, both as to methods of other clinics, and particularly as to that of Professor Rossi, the book is extremely valuable, and a notable propaganda for manipulative technique.

Die chirurgische Behandlung der Gehirntumoren: eine klinische Studie. By HERBERT OLIVECRONA. Royal 8vo. Pp. 344, with 228 illustrations. 1927. Berlin: Julius Springer. M. 27.

IT may be said of neurological surgery that the monographs which it has stimulated its devotees to write are amongst the most distinguished contributions which have seen the light in our time. Most have been confined to some particular pathological entity, but Dr. Herbert Olivecrona, of Stockholm, treats of intracranial tumours as a whole, and once more we have a book of unusual merit and one from which the surgeon working in this field can derive the greatest information. The book is essentially a personal record of four years' intensive experience. It illustrates well the fact that any man who takes up a special line for study will accumulate material that no one believed to exist. The point is well exemplified in the

Stockholm statistics. In the ten-year period 1912–21 only 31 cases of verified intracranial tumours were operated upon at the Serafimer-Krankenhaus, whilst in the four years 1922–26, during which time Olivecrona had full charge of a neurological service, there were 105 cases of tumours or tumour-suspects, of which 75 were verified (41 gliomas, 11 meningiomas, 12 neurinomas, 2 tuberculomas, and so forth). In a number of detailed tables the reader will find interesting analyses, but above all he will be interested and instructed by the admirable detailed descriptions of each and every one of the cases. The same plan is followed for all patients; first a short general history, then general pressure symptoms and localizing symptoms and signs, followed by the X-ray report and a complete account of the surgical treatment.

The clinical histories and the neurological signs are briefly and concisely given; in no case is there a long dissertation on this side of the problem, the one which has most enchanted the English mind in the past. And yet one is told enough to appreciate the case fully and to enjoy the problem presented. Olivecrona has made use of the common surgical aids to diagnosis. Radiological help (if one may be permitted to enlist as 'surgical' a neutral art) he has had from Dr. Lysholm, and those familiar only with English and American technique will be delighted with the revelation of yet another superb technician, strengthening Stockholm's claim to pre-eminence in the X-ray world.

The author used exploratory ventricular puncture in 13 cases, ventriculography in 14, and encephalography (lumbar air injection) in 4. The last produced the severest reactions, but the deaths, of which there were 2, followed ventricular injection—one a case of cerebral metastatic carcinoma, the second a deep glioma.

Olivecrona has made direct attacks upon all his tumours and has extirpated as many as possible. His mortality for decompression for gliomas is high: 7 deaths out of 9 cases of subtemporal, and 1 death in 2 suboccipital decompressions. Of 10 exposures and decompressions 5 died, whilst of 8 extirpations or treatment of cysts only 2 died. Thus the mortality varied inversely with the amount that was done, from which one infers that it was not the operation itself which brought a high mortality in its train, but the advanced nature of the case. The more benign cystic gliomas without underspread reactionary œdema are very favourable; others will succumb with the slightest interference, especially perhaps if the decompression cannot be placed accurately over the tumour. Œdema is unquestionably the bugbear of the glioma group, and is more important than hæmorrhage into the tumour, not because the latter does not occur but because it is certainly rarer. These arguments are pointed by an analysis of the general clinical condition of the patient. At the time of operation 7 were in coma, and 6 of them died; 10 had advanced generalized pressure, and 6 died; 11 showed only slight pressure changes, and of these only 3 died. Objection may be raised to the fact that the gliomas have not been well differentiated histologically, and a retrospect over each case from this viewpoint would have enhanced the already great importance of the book. Olivecrona's relatively large group of acoustic neuromas he owes to early recognition of the syndrome by his colleagues and himself; 3 of the 9 examples had no sign of choked disc. In general, 'choke' was present in 86 per cent of his verified tumour cases.

The author gives a full account of his operative methods, which do not differ in any essential point from those in general use amongst specialists to-day; but those eager to learn will find things of interest, and often something to admire, in the complete account which is given of the treatment of each one of the cases. Olivecrona uses local anæsthesia for choice, and does not hesitate to break off the operation and continue it in two or three days (not the classical two or three weeks which was the custom some years ago) if the patient is well, or more particularly if he is going downhill and something useful remains to be done. We are learning that a life may sometimes be saved by a bold second intervention, however recently an operation may have been performed. He has unquestionably risked some cases in which little or nothing could be hoped for—his comatose glioma patients and the angiomas are cases in point—but the critic's task is easier than the surgeon's.

The monograph is very well produced and excellently illustrated. All the post-mortem specimens have been hardened by injection of formalin before removal, the method on which Pierre Marie used so strongly to insist. Publication has been made possible by grants from special funds in Upsala and Stockholm, and these have been well applied in giving us so frank and illuminating a piece of work.

Pyelography: its History, Technique, Uses, and Dangers. By ALEX. E. ROCHE, M.A., M.D. (Camb.), F.R.C.S. Eng., Chief Assistant to a Surgical Unit, St. Bartholomew's Hospital. With an Introduction by Sir JOHN THOMSON-WALKER, O.B.E. Demy 8vo. Pp. 118 + xiv, with 16 plates. 1927. London: H. K. Lewis & Co. Ltd. 9s. net.

THIS is an excellent monograph, well and clearly written, and published in a most attractive and handy form; the author has not only studied the subject of pyelography with great care, but has been most assiduous in his attempt to present the value of this method of diagnosis with impartiality.

In his introduction to the work, Sir John Thomson-Walker lays stress on three points: firstly, that only those who are expert in cystoscopy and ureteral catheterization should undertake the making of pyelograms; secondly, the necessity of their interpretation by the experienced eye; thirdly, the limitation of this method to those cases in which the information desired could not be obtained by less severe methods of examination. Whilst we should all agree with the third point, it seems a little difficult, if the first two rules are strictly observed, to see how pyelography is to be carried on by future generations of urologists.

There is a good account of the history of pyelography, followed by a detailed account of the technique. We note that the author is in favour of 12 per cent sodium iodide as the opaque medium, and that this is injected by a syringe until the first appearance of discomfort in the loin. In this connection, emphasis is laid on the fact that in cases of hydronephrosis the pain due to distention of the pelvis is often absent, and that this may lead to a rupture of the thinned and distended wall.

The whole book is admirably written, and full of information. It is singularly free from printers' errors, though we observe that the letterpress explanatory of Plate 15 has been inserted upside down.

Amongst the contra-indications to the use of this method, we are surprised to find 'age' mentioned; this is surely too universal a complaint, and we are left uncertain whether the author means old age, or extreme youth, or both.

There is an index to the book and, under the heading of 'Authorities Consulted', an excellent bibliography; this is placed at the end of the work and is not too long.

La Pyéloscopie. By Professor F. LEGUEU, Clinique Urologique de Necker; BERNARD FEY, Chef de Clinique; and PIERRE TRUCHOT, Chef de Laboratoire de Radiologie. Large 4to. Pp. 111, with 36 plates. 1927. Paris: Editions Médicales, Norbert Maloine. 9s.

THE authors of this admirable monograph have suggested a modification of the usual examination of the urinary tract by X rays and an opaque solution; they point out that no surgeon is content nowadays with a mere X-ray photograph of the stomach and duodenum, and that a report from the skiagrapher on his observation of the parts as seen under the screen is always asked for; they have devised an ingenious technique for this purpose and have used the method at the Necker Hospital for three years; during this period they have examined over a thousand cases. This book gives the technique they advise for the examination under the screen of the ureter, pelvis, and calices; the larger part of the work is devoted to the results of their researches into the physiology and pathology of these parts.

Under the heading of technique we note that the authors consider it important to have the room for cystoscopy and that for X rays close to one another; that after the passage of the ureteral catheter the opaque medium (sodium iodide 30 per cent) is injected through a syringe; that this should be injected slowly until the pelvis and calices are clearly outlined on the screen, but that the injection should

stop before the patient feels any pain in the loin : otherwise, the pelvic muscle goes into spasm or shows irregular and violent contractions, in either case vitiating the findings ; and, finally, that they withdraw the catheter after filling the pelvis. They point out that the catheter, which must be opaque to the X rays, should only just enter the pelvis ; if pushed too far it may engage in the uppermost calix, which a very small amount of fluid will suffice to distend and so start these undesirable contractions. They also emphasize the importance of waiting until the observers' eyes are thoroughly accustomed to the dim light.

The only part of the apparatus which is not simple and inexpensive is the camera which they use in conjunction with the screening, so that, at any given moment, a plate may be exposed and a permanent record obtained ; although this was obviously necessary for the purpose of the present work, we imagine that a tracing of the condition found would answer in ordinary routine work.

If, as sometimes happens, the catheter is arrested at some point in its passage up the ureter, the patient is taken to the X-ray room and opaque fluid is injected to ascertain the cause of the obstruction ; by this means they found that the arrest is due to a kinking of the ureter and can be overcome by pushing up the kidney ; in over a thousand cases they never found a single case of stricture of the ureter ; this seems to us to be an observation of considerable importance in view of the great difference of opinion amongst urologists as to the frequency of this condition.

They state that if the catheter is left in the ureter with its end anywhere short of the pyelo-ureteric junction, the fluid will, as a rule, flow back along the side of the catheter, and that it is only in cases of atonic ureter that a pyelogram can be obtained under such conditions ; this statement will come as a surprise to many urologists, and would seem to make the X-ray diagnosis of ureter fissus more difficult than ever.

The authors describe a 'ureteral bulb' which they compare with the duodenal cap ; to this they ascribe a good deal of importance, and they consider that the X-ray appearances seen here are a criterion of the motility of the pelvic musculature. It is situated at the junction of the pelvis with the ureter, and normally a drop can be seen to form here every three to seven seconds ; after reading their account of the formation and behaviour of this drop, one is irresistibly reminded of the drop that forms at the outer-world end of a ureteral catheter, and one wonders whether too much importance is not attached to the ureteral bulb ; however, they attach great significance to the careful observation of this bulb, which they think is the visible expression of a sphincter at that level, and they give in detail the modifications undergone by this bulb in disease ; in fact, they have observed both retention and incontinence in this region.

The book is full of original observations which will be of the greatest interest not only to urologists and general surgeons but to physiologists and pathologists ; we will try to pick out a few striking examples.

In cases of a stone lying free in the pelvis, an incomplete evacuation of the pelvis was almost always noted ; this is not due, as generally taught, to mechanical obstruction, but to spasm or atony of the pelvic muscle. Incomplete evacuation was also noted in some, though not in all, cases of movable kidney ; but this, though accompanied by a kink in the ureter, is not caused by the kink, for the arrest occurs not at the kink but always at the level of the pelvis ; a similar condition obtains in cases of obstruction by an abnormal renal vessel, for, though the evacuation is incomplete, the arrest again occurs at the pelvis and not at the level of the vessel, which is beautifully shown as a transverse filling defect.

In all these three examples, the authors consider that the retention is not mechanical but is due to the presence of what they call an 'épine irritative', a picturesque wording for an exciting cause ; this they think suffices to throw the pelvic muscle into spasm or to cause atony if sufficiently prolonged.

The book is well and clearly written, the pyelograms are excellently reproduced, and the legends describing the latter are also given in English, Spanish, Italian, and German. We offer our hearty congratulations to the authors, and strongly recommend our readers who are interested in the subject of kidney surgery to study this stimulating monograph.